AD-A044 630 LETTERMAN ARMY MEDICAL CENTER SAN FRANCISCO CALIF F/G 6/5
AMBULATORY INTERNAL MEDICINE CARE SYMPOSIUM, PRESENT CONCEPTS I--ETC(U)
1977 T J EGAN, N Z SANDERS, C E SWEE

UNCLASSIFIED NL

1 OF 2
AD A044630

10
B.S

6

AMBULATORY INTERNAL MEDICINE CARE SYMPOSIUM, PRESENT CONCEPTS IN INTERNAL MEDICINE.

10

LTC Thomas J. Egan, [redacted], Nina Z. Sanders, [redacted] and Cathleen E. Swee, B.A.

H. L. Eichner
J. A. Feagins

Letterman Army Medical Center
Presidio of San Francisco, CA 94129

11 1977

12 122p.

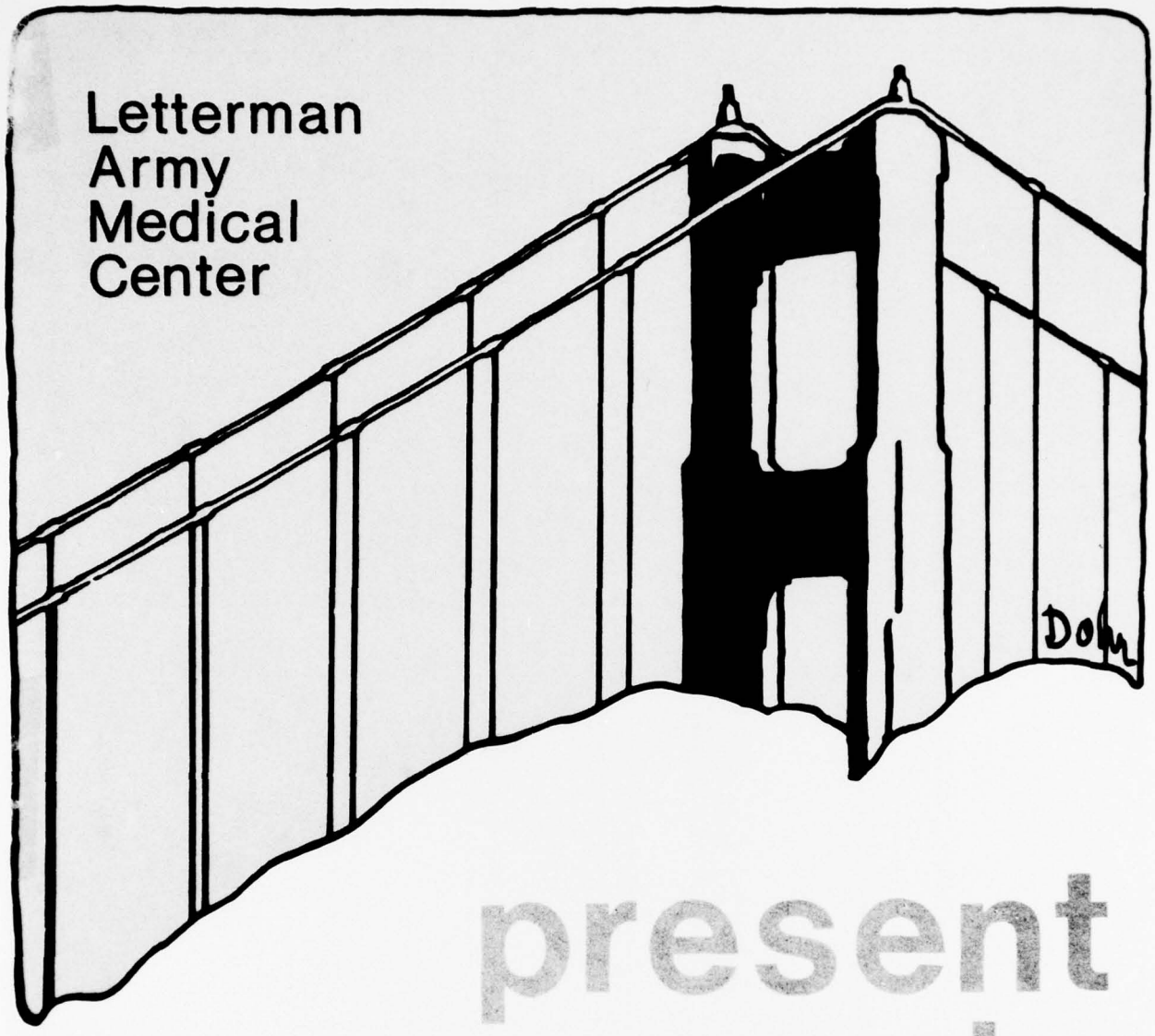
Ambulatory Internal Medicine Care Symposium

Approved for public release;
distribution unlimited.

prepared for
Letterman Army Medical Center
Presidio of San Francisco, CA 94129

408 172

AMBULATORY INTERNAL MEDICINE CARE SYMPOSIUM present concepts in internal medicine summer 1977



present concepts

in Internal Medicine

AMBULATORY INTERNAL MEDICINE CARE SYMPOSIUM, SUMMER 1977

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE *(and Subtitle)* Present Concepts in Internal Medicine Ambulatory Internal Medicine Care Symposium, Summer 1977		5. TYPE OF REPORT & PERIOD COVERED Medical Symposium - Summer 1977 6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) TJ Egan, NZ Sanders, CE Swee (eds); HL Eichner; JA Feagin,Jr.; AL Franger; PJ Killian; W Nash and DT Baumann; RB Odom; R Ogburn; JP Schmidt; DH See; FL Wergeland, Jr.		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS Department of Medicine (AFZM-MDM) Letterman Army Medical Center Presidio of San Francisco, CA 94129		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS AFZM-MDZBTE, Letterman Army Medical Center Presidio of San Francisco, CA 94129		12. REPORT DATE Summer 1977 13. NUMBER OF PAGES 120
14. MONITORING AGENCY NAME & ADDRESS*(if different from Controlling Office)* Same		15. SECURITY CLASS. *(of this report)* Unclassified 15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT *(of this Report)*

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT *(of the abstract entered in Block 20, if different from Report)*

NA

18. SUPPLEMENTARY NOTES

19. KEY WORDS *(Continue on reverse side if necessary and identify by block number)*

Psychosomatic Medicine; Sinusitis; Eye Diseases; Vaginitis; Dermatitis; Thyroid Function Tests; Polyps; Heat; Infectious Mononucleosis; Shoulder

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)*

This symposium consists of 10 articles and 18 tables (for quick reference). It includes articles on using a behavioral approach to psychosomatic complaints; sinusitis; the red eye; management of vaginal discharge; common dermatologic problems; the testing of thyroid functions; colorectal polyps; the rational use of therapeutic heat; infectious mononucleosis; and an article on shoulder pain.

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE *(When Data Entered)*

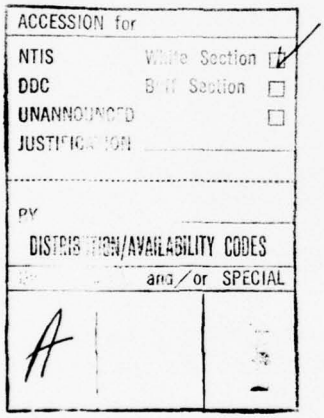
PRESENT CONCEPTS
IN INTERNAL MEDICINE

LTC Thomas J. Egan, MC
Guest Editor

Nina Z. Sanders, B.A.
Technical Publications Editor

Cathleen E. Swee, B.A.
Assistant Editor

LETTERMAN ARMY MEDICAL CENTER
Presidio of San Francisco, California 94129
Ambulatory Internal Medicine Care Symposium, Summer 1977



PRESENT CONCEPTS IN INTERNAL MEDICINE

Ambulatory Internal Medicine Care Symposium, Summer 1977

CONTENTS

Department of Medicine
LETTERMAN ARMY MEDICAL CENTER
Presidio of San Francisco, California 94129

FORTHCOMING SYMPOSIA

GASTROENTEROLOGY

DERMATOLOGY

HEMATOLOGY/ONCOLOGY

INFECTIOUS DISEASES

ENDOSCOPY

FORWARD

Because of the ever-increasing specialization on the part of the present day physician and hence the gradual disappearance of the general practitioner from the medical scene, the responsibility of adult primary care has shifted at least in part to the general internist.

The American Board of Internal Medicine has stressed in recent years that common dermatology, gynecology and otolaryngology disorders which have traditionally been managed by the general practitioner are now within the newly defined limits of the field of Internal Medicine and, furthermore, some teaching in these areas is a requirement of each Internal Medicine training program.

In this issue of *Present Concepts in Internal Medicine,* an attempt is made to cover some common disorders which the internist will encounter in everyday office practice and which until only recently have been included under the heading of "Internal Medicine".

Hopefully, these articles will help to make the office and clinic practice more interesting to our physicians and, at the same time, provide some benefit in better patient management.

THOMAS J. EGAN, MD
Lieutenant Colonel, MC
Chief, Ambulatory Internal Medicine Service
Guest Editor

If you start to think about your physical or moral condition, you usually find that you are sick.

Johann Wolfgang von Goethe
(1749 to 1832)
Sprüche in Prosa
Pt. i, Bk 2

A BEHAVIORAL APPROACH TO PSYCHOSOMATIC COMPLAINT

CPT James P. Schmidt, MSC

One of the problems most frequently confronting the internist or general practitioner is the patient with a psychosomatic complaint; Lewis /1/ in 1952 concluded that 47 percent of all patients seen by physicians had a physical disorder caused in some part by a psychological disturbance. There is no evidence to suggest that the prevalence of psychosomatic complaints has decreased since that time. Indeed, there is subjective evidence that it has *increased*, with many physicians stating that one half to two thirds of their patients have psychosomatic complaints. The problem is intensified by the relative lack of training of both mental health professionals and physicians in the management of such patients, and by the assumption that they are difficult or impossible to treat. This paper provides a behavioral model for the understanding of psychosomatic disorders, a discussion of the factors useful in their classification, and a series of specific suggestions for their basic management.

I shall first define the terms "psychosomatic complaint" and "behavorial approach", and present an overview of the traditional theory of psychosomatic illness and the research on the topic.

DEFINITION OF PSYCHOSOMATIC COMPLAINT

While the term "psychosomatic disorder", or as it is more recently termed, "psychophysiologic disorder", has a relatively narrow formal definition /2/, in general usage among physicians it commonly refers to four related phenomena. (It is important to note that this definition has

little or no known etiologic implications from the *behavorial* viewpoint; rather, it represents a classification of presenting complaints.)

1. *The Chronic Complaint Patient.* This patient shows chronic concern for his health and frequents the physician's office with minor complaints which he fears reflect a serious condition. In psychiatric literature, the patient is said to have a "hypochondriacal neurosis". /2/

2. *The Overreactive Patient.* While he does have a diagnosed medical disorder, this patient overreacts to the disorder either by interpreting it as something far more serious, or by allowing it to unduly alter his lifestyle.

3. *The Nonexistent-Syndrome Patient.* The patient who has definite physical symptoms with no physical basis differs from the chronic complaint patient in that the complaints tend to be specific and stable across time. This patient most closely fits the diagnostic category of "hysterical neurosis". /2/

4. *The Stress-Induced Syndrome Patient.* The stress-induced syndrome patient meets the technical definition of psychophysiologic reaction: presence of a real physical illness which is triggered, maintained, or intensified by stress. Conditions often included in this group are migraine, neurodermatosis, functional back pain, peptic ulcer, impotence, and hypertension. /2/

In addition to the above phenomena, many patients with psychosomatic complaints have the added problem of chronic drug use. A high proportion of these patients are on some form of minor tranquilizing or sleep-inducing medication.

DEFINITION OF BEHAVIORAL APPROACH

Behavorial approach or "behavior therapy" includes a variety of techniques which address a myriad of human problems;

the common thread among these techniques is their derivation from, and dependence upon, scientific research. Thus, while models such as psychoanalysis are primarily theoretically derived and evolved, behavior therapy has drawn most of its techniques from three areas of research: learning theory (the effects of reward and punishment); cognitive psychology (how thoughts influence feelings and behavior); and social psychology (the effects of environment on learning patterns of individuals). The roots and applications of behavior therapy are described by Lazarus /3/, and Goldfried and Davison. /4/ Despite its recent advent, several exploratory applications of behavior therapy have been made to psychosomatic problems, and at least two books have been written on this topic. /5,6/

THE TRADITIONAL MODEL OF PSYCHOSOMATIC DISORDER

The traditional model of psychosomatic disorder is that the physical complaints are symbolic representations of one or more personality factors. Dunbar /7/ contends that these factors are tied to personality type, while Alexander and French /8/ believe they reflect a specific emotion or conflict. In discussing the latter, more popular view, these authors state that establishing the specific relationship requires long, refined, systematic investigations with the help of the psychoanalytic technique. Examination of the major psychiatric textbook /9/ reveals that current psychiatric thought continues to place major emphasis on psychodynamic formulations, elaborating on them rather than seeking alternatives to them.

MAJOR RESEARCH FINDINGS

While most *theory* focuses on psychological factors, much of the *research* focuses on the physiological aspects of the psychosomatic complaint. The research falls into four major

areas: stress reactions, stress reactivity, progressive advances in biofeedback technology, and attitude-specific responses.

Stress Reactions

This research by Selye /10,11/ reveals important facts about the psychogenesis of psychosomatic complaints: (1) there is a physiological response pattern to external stress termed the "General Adaptation Syndrome" (GAS) which follows a predictable course; and (2) extended periods of time in the GAS produce dysfunction in the organism. Selye's research constitutes some of the first empirical evidence that environmental events can create physiological reactions leading to demonstrable physical damage to the organism. He further shows this mechanism to be common across species and not isolated to humans.

Stress Reactivity

Two types of research focus on the differences among individuals in terms of stress reactivity: (1) The concept of "autonomic tuning", popularized by Gellhorn and Loofbourrow /12/ which states that an individual may produce a sympathetic or parasympathetic response to a given stimulus, depending upon the "tuning" of the hypothalamus or the predisposed potential to respond in one direction or the other. (2) The research dealing with reactivity levels of infants to various stimuli /13-16/ which shows consistent individual differences in magnitude, speed, and duration of cardiac changes following stimulation, and demonstrates physiologically what others have observed behaviorally. /17/ It may be concluded that there apparently are genetically-determined differential levels and styles of reactivity.

Advances in Biofeedback Technology

Research indicates that physiological responses previously classified as involuntary (skin temperature, blood

pressure, heart rate, and electrical activity in the brain) can be shaped and conditioned by the same laws and principles that determine other learned behavior. /18,19/ This literature shows that bodily functions can be controlled by learning, that organ systems can be taught to respond differentially, and that relearning approaches can successfully eliminate psychosomatic problems. Therapeutic literature /20,21/ indicates that tension headaches and other muscle tension disorders are treatable by biofeedback, and that there are promising findings in treatment of conditions such as cardiac arrhythmias, hypertension, seizure disorders, asthma, and peptic ulcers.

Attitude–Specific Responses

This research, conducted primarily by Graham et al /22-26/, suggests that specific attitudes toward stressful situations are closely correlated with specific psychosomatic reactions. For example, patients who react to stress with feelings of helplessness tend to develop hives, while those with feelings of aggression tend to develop Raynaud's disease, and those who feel threatened develop essential hypertension. In a study of 128 patients /22/, common attitudes of stress were shared by patients with syndromes of urticaria, eczema, cold hands, vasomotor rhinitis, asthma, diarrhea, constipation, nausea, duodenal ulcer, arterial hypertension, migraine, and low back pain.

The research clearly demonstrates that different attitudes toward stress can predispose individuals to develop different types of psychosomatic complaints. While it supports the *psychoanalytic* hypothesis that there is a relationship between conflict and symptom, it contradicts the *analytic* hypothesis that these attitudes are deeply hidden and can be reached only through extensive psychoanalysis. In fact, treatment via direct identification and disputation of attitudes has been effective in eliminating these attitudes. /27,28/ An approach in which the therapist teaches the patient to recognize his own self-damaging statements has been an effective technique in treatment of psychological distress. /4,29/

In summary, it has been demonstrated that all individuals have a physiological tendency to react to stress in a

systematic fashion. This tendency varies according to genetic predisposition; its specific aspects can be modified by learned experiences. The specific type of attitude toward stress influences the type of stress reaction which results.

BEHAVIORAL CLASSIFICATION OF PSYCHOSOMATIC COMPLAINTS

Five general factors appear to emerge as consistently important in the evaluation and management of psychosomatic patients:

1. *Sensitivity to Bodily Cues.* Awareness of internal bodily state varies with individuals. Some people are highly aware of their internal state; others are not. Individuals on either extreme of the continuum present a higher potential for psychosomatic complaints than do those in the middle range. Individuals highly aware of their bodily state often tend to interpret normal bodily changes and fluctuations as illness. It should be emphasized that it is not *awareness of change which is maladaptive*, but rather the *meaning* attributed to that change. The complaints of individuals who lack awareness of their bodily state are apt to be of the "pure" psychosomatic type; they develop psychosomatic complaints because they do not notice the signs of stress and body tension and thus take no steps to reduce or eliminate them. Consequently, they remain in an activated state until reaching the GAS exhaustion stage. Only when physical symptoms become acute do they seek medical attention.

2. *Internal versus External Source of Stress.* While in all cases stress has both external (environmental) and internal (cognitive) components, one is usually stronger than the other. In treating *external* stress, environmental manipulation may be appropriate. Thus, an individual who submits to a form of stress such as rush hour traffic might find a way to avoid or alter the situation. Cognitive restructuring may be the therapy of choice in cases involving *internal* stress. This does not necessarily imply that a behavior therapist must become involved in treatment; many basic techniques can be successfully implemented by the physician. Some of these are discussed later in the paper.

3. *Chronic versus Acute Problem.* This dimension is a critical one in psychosomatic patients. Patients with chronic psychosomatic problems typically require comprehensive therapy because their symptoms have a long history, and because the symptoms tend to be deeply embedded in their overall lifestyle. Thus, while the patient with a minor or *acute* psychosomatic complaint may be successfully treated by a physician, the patient with a *chronic* complaint may ultimately require the expertise of a behavior therapist.

4. *Presence of Reinforcement.* There are two basic types of reinforcement: attainment of a reward, and avoidance of a punishment. For example, a patient may be ill because he obtains attention from family members (reward), or because he can avoid having to do an unpleasant job (avoidance of punishment), or perhaps a combination of the two. In cases where reinforcement is detected, it may be possible to help the patient find ways to eliminate the reinforcing qualities and thus decrease the likelihood of continuation. For example, the chronic pain patient may find that avoidance of work is reinforcement for pain. If he can force himself to work, however, the pain diminishes. It must be strongly emphasized that reinforcing the symptom does *not* imply that the patient is purposely seeking reinforcement, nor that he is *aware* of the positive consequences; *usually the reinforcement is completely out of his awareness.* Thus, the physician determines the reinforcement by asking how the problem has altered the patient's lifestyle.

5. *Functions of the Symptom.* Frequently the psychosomatic symptom serves a behavioral function for the patient. For example, the patient with low back pain may, without awareness, use this pain as a social tool by discussing it with others or to manage difficult interpersonal relations. In this instance, it may be helpful to offer the patient an alternative strategy such as assertiveness training. /32/ Likewise, other forms of social skills training such as time budgeting, money management, and conversation skills may be beneficial for psychosomatic patients who place heavy reliance on their symptoms as a social vehicle.

BEHAVIORAL TECHNIQUES FOR PSYCHOSOMATIC COMPLAINTS

The following behavioral techniques are commonly used strategies which have been modified or adapted for use with patients manifesting psychosomatic complaints; some of the techniques have been devised by the author. All have been applied to psychosomatic problems by the author with some degree of success*. Key criteria in choosing techniques to be used are the amount of time and expertise required for their application. One weakness of the psychodynamic approach is that it offers virtually no direct strategies in the management of psychosomatic complaints, suggesting instead long-term psychodynamic therapy as the only appropriate treatment. While these techniques do not represent a solution to *all* psychosomatic problems, or substitute for a good doctor-patient relationship, it is hoped that they will offer the physician some ways to aid in the management of acute and less severe psychosomatic complaints.

Rapid Relaxation Technique

A modification of Maultsby's "instant better feeling maneuver" /31/, this technique serves a dual function: first, it demonstrates to the patient the degree of his tension and raises his awareness of bodily state; and second, it provides a method of reducing the tension level without medication. The technique is demonstrated to the patient in the office and he is then encouraged to practice it at home. Briefly, the technique is as follows: the patient closes his eyes and draws and exhales five deep breaths, pausing 6 to 10 seconds between each breath. During each of the four pauses, the patient concentrates on relaxing a different muscle group: first, the facial muscles; second, the

*The author would appreciate receiving feedback from readers about the successes and failures of these techniques as they apply them. These comments can be addressed to him at Box 603, Letterman Army Medical Center, Presidio of San Francisco, CA 94129 or at telephone 415-561-5115.

muscles of the chest and abdomen; third, the muscles of the arms; and fourth, the muscles of the legs. After the fifth breath, he opens his eyes and resumes normal breathing. The entire procedure takes only one minute, and gives the patient a sense of relaxation in sharp contrast to his earlier sensations. With practice, the patient can attain a relaxed state in a matter of seconds. This technique is useful with patients who are unable to differentiate bodily state, and is also a useful first step with those who complain of chronic tension.

Defining Situational Determinants Technique

Both diagnostic and therapeutic, this technique constitutes a standard component of all behavioral interviews. The patient is asked to give a detailed description of several symptomatic episodes, focusing on what was going on just before their onset. As he describes three or four situations, it may be fairly easy to discern common events which preceded the situations. The patient is then given a clear strategy for reducing the problem: avoidance, insofar as possible, of those situations which trigger the episodes. It must be noted that the term "situation" as used here includes self-statements about the patient's internal and external situation. Thus, a description of the circumstances leading up to the episode should include the patient's thoughts at the time.

Eliciting Natural Coping Strategies Technique

Typically, the patient has his own techniques which are at least partially successful in reducing the symptomatology. Discussing these techniques with the patient has several advantages: (1) it illustrates how behavior (such as walking on the beach to get rid of a headache) can be used to change the symptom; (2) it usually raises to a higher level of awareness some of the automatic techniques the patient has been using; and (3) the physician's support of these methods gives the patient a sense of validity and value.

Education Technique

A technique which is highly effective is simple education about how the autonomic nervous system works, what the physiological signs of arousal are, and how they relate to the particular symptom pattern of the patient. A clear description of phenomena such as the effects of increased adrenalin output offers the patient a structure to understand his symptoms, thereby reducing the common fear that signs of autonomic arousal are symptoms of a more serious condition. Additionally, it heightens the patient's sensitivity about signs to watch for in monitoring his own stress levels.

Prediction of Symptoms Technique

This technique is closely related to the previous one and has the same functions. When a patient begins to describe autonomic arousal symptoms, the physician stops him and describes the other symptoms he is likely to experience. Thus, when the patient describes shortness of breath, the physician might interject that sweaty palms, rapid heart beat, and trembling are also common. This is reassuring in that it shows that the physician does sense what the patient is experiencing, and that it is indeed a real reaction pattern. Many patients believe that the term "psychosomatic" is reserved for cases where the doctor does not know what is wrong, and so assumes it is "all in my head". This belief, one of the most potentially destructive misconceptions a patient can have, may be lessened or eliminated through re-education and empathic support by the physician.

Elimination of Physical Causes Technique

Typically, the physician will make an exhaustive physical examination prior to diagnosing the condition as a psychosomatic one. While appropriate, this technique may be either damaging or therapeutic, depending upon how it is handled.

If the patient feels that the doctor has failed to find anything and is "writing him off", it will prove anti-therapeutic. If, however, the physician explains to the patient beforehand that the condition is probably stress-induced and that the examination is routinely done to rule out any physical cause, the examination is likely to be therapeutic.

Reattribution Technique

This technique, which consists of offering the patient new or alternative explanations for observed symptoms, is similar to the educational approach. One of its applications consists of restating the patient's symptoms as behaviors. Thus, if the patient says, "I have shortness of breath", the physician may reattribute it as "You tend to breath too rapidly and shallowly." This procedure serves the dual function of removing the fearful connotation from the phenomenon, and at the same time, giving the patient an increased sense of control over it. Rephrasing "pain" as "discomfort" and "inability" as "difficulty" are other ways in which reattribution may help the patient gain a perspective on his symptoms.

Bibliotherapy Technique

Frequently, patients will benefit from readings which describe their syndromes and suggest ways of coping with them. I have found the following books useful: Stress, by Ogden Tanner (New York: Time-Life Books, 1976) which describes stress reactions; A New Guide to Rational Living, by Albert Ellis and Robert Harper (North Hollywood: Wilshire, 1975) and Help Yourself to Happiness Through Rational Self-Counseling by Maxie Maultsby (New York: Institute for Rational Living, 1975), which describe cognitive restructuring; and The Relaxation Response, by Herbert Benson (New York: Avon, 1976) which describes relaxation and its effects.

Chart Review Technique

Reviewing the medical chart with the patient may be helpful in reducing anxiety. Patients are frequently told a variety of things by various physicians. Many patients retain only fragments of these conversations and worry unnecessarily over conditions once mentioned as "possible" and then later ruled out. Thus, over a period of time, some patients become anxious and upset over their health, simply because of misunderstandings. This situation may be remedied by reviewing the medical record, explaining each entry to the patient, and summing up the information in a few well-selected sentences. I have found this method to be especially effective with patients who have a limited command of the English language.

Cognitive Restructuring Technique

Cognitive restructuring requires a high level of training; however, even without such training, the physician can often use it with good results. It has been my experience that many patients tend to use one of three maladaptive cognitive tendencies. The first of these is the tendency to think dichotomously. That is, something either "is" or it "is not"; the patient is either "sick" or "well", and the illness is either "organic" or "all in my head". It is helpful for the patient to realize that this is not necessarily so; but rather that illness is on a continuum, along which the patient moves. The patient should be informed that an illness can have both physical and psychological causative factors.

A second common tendency is that of forming illusory correlations. People tend to remember unusual or sensational events rather than the common ones. Thus, they clearly remember the individual who got breast cancer after having an x-ray, but they forget the hundreds of others who had x-rays and did not develop cancer. Such beliefs may be changed by pointing out to the patient instances where it did *not* happen, and by citing reassuring statistics.

The third common tendency is to escalate situations, thereby turning unknown situations into fearful ones, and irritations into anger. Frequently, the physician can demonstrate to the patient how a situation can be escalated into a crisis by what the patient thinks to himself and how that perceived crisis can trigger the GAS. This technique must be done supportively and sympathetically in order to achieve an alliance with the patient.

CONCLUSION

In conclusion, a few comments about the general management of psychosomatic problems are in order. Patients with psychosomatic complaints are often annoying and bothersome to the physician; however, angry reactions, lectures, or dismissal of their symptoms as being "all in your head" is seldom therapeutic. This is not to imply that such patients should be pampered or encouraged in their complaints. Rather, the etiologic nature of their problems, together with recommendations for more adaptive responses, are best discussed with them in a nonevaluative attitude. Frequently the calm reassurance by the physician that the symptoms are indeed real is all that is needed; in other cases, identification of stress-inducers and implementation of simple strategies are sufficient. Some patients, however, will still require the expertise of a mental health professional. In any case, the goal of this paper will be met if it helps the reader find ways to reduce his own anxiety and anger in dealing with psychosomatic patients, and provides some new techniques which can effectively be applied in their treatment.

ACKNOWLEDGEMENTS

The author gratefully acknowledges the valuable additions to this paper in the form of editorial suggestions from CPT D. Kris Picklesimer, MSC, CPT Thomas K. Sims, MSC, and CPT Gerald W. Urbancik, MSC.

References

1. Lewis BI: Psychosomatic disorders and nonpsychiatrists. JAMA 150:776-780, 1952.
2. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders. Washington DC, American Psychiatric Association, 1968.
3. Lazarus AA: Behavior Therapy and Beyond. New York, McGraw-Hill, 1971.
4. Goldfried MR, Davison GC: Clinical Behavior Therapy. New York, Holt, Rinehart and Winston, 1976.
5. Lachman SJ: Psychosomatic Disorders. New York, Wiley, 1972.
6. Katz R, Zlutnick S: Behavior Therapy and Health Care. Elsmford, New York, Pergamon Press, 1975.
7. Dunbar F: Psychosomatic Diagnoses. New York, Hoeber-Harper, 1943.
8. Alexander F, French TM: Studies in Psychosomatic Medicine. New York, Ronald, 1948.
9. Freedman AM, Kaplan HI, Sadock BJ: Comprehensive Textbook of Psychiatry II. Baltimore, Williams & Wilkins, 1975.
10. Selye H: The Stress of Life. London, Longmans, 1957.
11. Selye H: Stress. Psychology Today. 3:24-26, 1969.
12. Gellhorn E, Loofbourrow GN: Emotions and Emotional Disorders. New York, Hoeber, 1963.
13. Lipton EL, Steinschneider A, Richmond JB: Autonomic Function in the Neonate II. Psychosom Med 22:57-65, 1960.
14. Lipton EL, Steinschneider A, Richmond JB: Autonomic Function in the Neonate III. Psychosom Med 23:461-471, 1961.
15. Lipton EL, Steinschneider A, Richmond JB: Autonomic Function in the Neonate IV. Psychosom Med 23:472-484, 1961.

16. Richmond JB, Lipton EL, Steinschneider A: Automonic Function in the Neonate V. Psychosom Med 24:66-74, 1962.
17. Thomas A, Birch HG, Ches S, et al: Behavioral Individuality in Early Childhood. New York, University Press, 1965.
18. Miller NE: Learning of visceral and glandular responses. Science 163:434-445, 1969.
19. Peper E: Frontiers in Clinical Biofeedback. In Birk L (ed): Seminars in Psychiatry, New York, Grune & Stratton, 1973, vol 5.
20. Schwartz GE: Biofeedback as therapy. Am Psychol. 28:666-673, 1973.
21. Blanchard EB, Young LD: Clinical applications of biofeedback training. Arch Gen Psychiatry 30:573-589, 1974.
22. Grace WJ, Graham DT: Relationship of specific attitudes and emotions to certain bodily diseases. Psychosom Med 14:243-251, 1952.
23. Graham DT, Kabler JD, Graham FK: Physiological response to the suggestion of attitudes specific for hives and hypertension. Psychosom Med 24:159, 1962.
24. Graham DT, Stern JA, Winokur G: Experimental Investigation of the specificity of attitude hypothesis in psychosomatic disease. Psychosom Med 20:446-457, 1958.
25. Graham DT: Some research on psychophysiologic specificity and its relation to psychosomatic disease, in Roessler R, Green NS (eds): Physiological Correlates of Psychological Disorder. Madison, University of Wisconsin Press, 1962.
26. Maultsby MC, Graham DT: Controlled study of effect psychotherapy on self-reported maladaptive traits, anxiety scores and psychosomatic disease attitudes. J Psychiatr Res 10:121-132, 1974.
27. Ellis A: Reason and Emotion in Psychotherapy. New York, Lyle Stuart, 1962.
28. Ellis A: Growth Through Reason. Palo Alto, Science and Behavior Books, 1971.
29. Maultsby MC, Gram JM: A Two-Year Followup Study of Rational Behavior Therapy. Presented at the 5th Annual Meeting of the Association for the Advancement of Behavior Therapy, 1971.

30. Gottlieb HJ, Hockersmith VW, Koller R, et al: A Successful Treatment Program for Chronic Back Pain Patients. Presented at American Psychological Association Convention, 1975.
31. Maultsby MC: Help Yourself to Happiness Through Rational Self-Counseling. New York, Institute for Rational Living, 1975.

"HEY, DOC, I GOT SINUS"

CPT William Nash, MC and LTC David T. Baumann, MC

It is estimated that over 13,000,000 Americans suffer from problems related to the nose and paranasal sinuses. This paper will discuss acute, subacute, and chronic sinusitis, allergic rhinitis, and vasomotor rhinitis, and try to help the physician understand, diagnose, and treat these illnesses.

ACUTE SINUSITIS

Acute sinusitis is an inflammation or infection of the mucosal lining of the sinus that occurs most frequently following upper respiratory infection, swimming, or diving. Symptoms, starting with a stuffy feeling in the nose and a slowly-developing pressure over the sinus, progress to signs of localized tenderness over the involved area, with an accompanying headache. With adequate treatment, more than 90 percent of the patients with acute sinusitis are cured. If sinusitis is neglected during the acute or subacute phase, or if recurrent attacks damage the mucosa, permanent tissue changes may take place. When the physician believes *irreversible* tissue change has occurred in the lining membrane of one or more of the paranasal sinuses, the diagnosis of chronic sinusitis is made.

Signs and Symptoms

At the onset of an attack of acute sinusitis, the patient may complain of a stuffy feeling in the nose and pressure over one of the sinuses. A feeling of generalized

malaise and headache may be present. Over the next 48 to 72 hours, the symptoms worsen until there is localized pain and tenderness over the involved sinus. The pain is usually more severe one to two hours after arising in the morning, and less severe in the afternoon and evening. When the maxillary sinus is involved, pain is localized over the cheek and associated teeth. The patient may present with a complaint of an abscessed tooth. To help differentiate between the two, the teeth may be tested by tapping them with a probe; with a diseased tooth, there is usually pain in only one tooth, whereas with maxillary sinusitis several may be involved.

The patient with ethmoiditis (perhaps the most frequently occurring type of acute sinusitis) usually presents with pain medial, deep to, or in the eye. The patient may also have anosmia. In infections of the sphenoid sinus the pain is deep, behind the eye, in the occiput, or sometimes referred to the vertex of the skull.

Pharyngitis is a common complaint with most sinusitis and is caused by irritation created by the purulent discharge. Look for a history of allergy in the patient presenting with acute sinusitis. Most rhinologists believe allergy to be the predominant and predisposing factor to sinusitis. /1/ *It should be emphasized that the nasal mucous* membranes are normally quite resistant to bacterial invasion. Any factors which impair the mucous blanket or ciliary activity will predispose the mucous membranes to infection; these factors include allergy, trauma, stress, exposure to toxic agents, temperature and humidity changes, and smoking.

Physical Examination

Physical examination of the patient with acute sinusitis should include palpation of the sinuses to elicit tenderness. Transillumination of the frontal and maxillary sinuses may reveal an opacity in one of them. Vital signs may show a low grade temperature of 37.2° to 37.5° C, or the temperature may be subnormal. The nasal mucosa on the involved side is often hyperemic and edematous and a purulent discharge is evident in most patients. The purulent discharge may best be seen by shrinking the edematous mucosa with ¼ percent phenylephrine hydrochloride USP (Neosynephrine® Hydrochloride) and then using a nasal speculum to examine the turbinates.

Laboratory and Radiographic Studies

Although laboratory and radiographic studies may be helpful in diagnosing acute sinusitis, they are not always necessary to establish the diagnosis. An allergy screening test is recommended because of the relationship between sinusitis and allergy. A complete blood count usually shows a leukopenia (white blood count 5,000 to 6,000); the reasons for this are not known. If it is determined that radiographic studies would aid in establishing the diagnosis, the following films should be included: (1) a Waters view to see the maxillary sinus; (2) a Caldwell view to visualize the frontal and ethmoidal sinuses; (3) a lateral view to aid in visualizing the sphenoidal sinuses and the posterior wall of the frontal sinuses; and (4) a submental vertex view to see the sphenoidal and ethomidal sinuses. It is important to realize that sinus radiographs are generally unreliable in diagnosing ethmoiditis and sinusitis in children because of the high incidence of false positive and false negative readings.

Treatment

The treatment of acute suppurative sinusitis is medical rather than surgical. The exception to the rule is when the natural ostium of the sinus is completely blocked, leading to empyema which usually requires surgical drainage. Codeine or a similar drug is necessary for pain; aspirin is not adequate. The application of wet heat and steam inhalation may be beneficial. In order to promote drainage, the nasal mucosa should be shrunk with normal saline nose drops and a nasal spray containing a vasoconstrictor such as 2 percent ephedrine or 0.25 percent Neosynephrine® . Decongestants by mouth are helpful, but anthihistamines should not be used unless allergy is a significant component. (NOTE: Shrinking agents result in deprivation of blood supply, followed by rebound swelling; their chronic use may lead to rhinitis medicamentosa). Antral irrigation for those patients whose sinus problems fail to

resolve quickly is also helpful. Although the value of antibiotics is questionable, we feel it is necessary to use them because they may help decrease the possible complications of acute sinusitis.

For treatment of ethmoiditis or sphenoiditis, direct evacuation is impossible and drainage can be obtained only by the Proetz displacement method. Displacement therapy should not be used during acute suppurative sinusitis, because it may spread the infection to other areas. Antibiotics are beneficial and should be selected to treat the cultured organism. Antihistamines may be helpful because persistent sinus problems often have an allergic component. The systemic administration of a steroid such as prednisone, 20 mg each day for three to five days, may also be helpful because of its anti-inflammatory and anti-allergic effects.

The Center of Disease Control has noted that the most common pathogens for paranasal sinus infections, in order of frequency, are: *Diplococcus pneumoniae; Group A Streptococcus; Staphlococcus aureus; Haemophilus influenzae;* the *Enterobacteriacae;* and *Pseudomonas*. However, there is evidence that *S. aureus* is twice as common as *D. pneumoniae* and definitely more common in acute maxillary sinusitis. /2/ *D. pneumoniae* and *Streptococcus* respond well to penicillin or erythromycin, the best all-around antibiotic for acute sinusitis. If the infection continues but the cultures remain negative, anaerobes and L-forms may possibly be causing the infection. Finally, aggravating factors such as smoking, spicy foods, ethyl alcohol, chilly weather, and allergic exposure should be avoided.

Prognosis and Complications

The patient with acute sinusitis will usually have discomfort and nasal discharge for three to four days, followed by slow resolution of the infection with complete relief occurring in 10 to 14 days. Complications usually depend on which sinus is involved. The most frequent complication of acute sinusitis is chronic sinusitis. Frequently, sinusitis causes occlusion of the sinus ostium and requires surgical drainage.

Ordinarily, any surgical procedure involving the bony walls of the sinus is contraindicated during the acute phase of sinusitis because osteomyelitis may occur. Occlusion of the ostium most frequently occurs with frontal sinusitis and, if not treated, can lead to osteomyelitis (Pott's Puffy sign) and meningitis, or a brain abscess. Ethmoiditis may lead to periorbital or orbital abscess. The most feared complication is that of cavernous sinus thrombosis which carries a 50 percent mortality. The infection generally travels from the nose to the angular vein and then to the cavernous sinus where septic thrombosis occurs. The signs and symptoms are sudden chills, fever, ocular paralysis beginning with one cranial nerve and finally involving all three nerves to the ocular muscles, periorbital edema and headache, going on to meningitis, intracranial hypertension, brain abscess, and disseminated septic thrombus.

SUBACUTE SINUSITIS

Some patients develop subacute suppurative sinusitis, which is essentially an acute sinusitis that fails to respond to treatment. The hallmark of subacute suppurative sinusitis is persistent purulent nasal discharge. The nose may remain stuffy but severe, local tenderness over the involved sinus will usually subside into a vague discomfort over the involved area. The patient may complain of feeling run down and of tiring easily. An unproductive cough, secondary to chronic pharyngeal irritation from the discharge, may also be present.

Physical Examination

Upon physical examination, one should look for persistence of pus in the nose. The involved sinus will not transilluminate, and radiographs will demonstrate changes in the sinus. There will be only slight discomfort on sinus palpation.

Treatment

In treatment of subacute suppurative sinusitis, generally no medications are needed to relieve the pain. The nasal mucosa should be shrunk with drops or vasoconstrictor sprays. Irrigation of the sinus should be carried out if it can be performed without damaging the ostium. Antral puncture for treatment of maxillary suppurative sinusitis is a beneficial procedure.

CHRONIC SINUSITIS

The patient with chronic suppurative sinusitis will present with purulent nasal discharge and chronic nasal congestion, or both. An increased incidence of nasal and sinus polyps may also occur in chronic sinusitis. Although the patient may complain of headache, it is important to realize that a frontal headache is more frequently caused by allergic or vasomotor rhinitis than by a chronic infection of the paranasal sinuses. For a diagnosis of chronic sinusitis, there must be a history of repeated sinus infections producing permanent mucosal damage, with the presence of pus in the sinus demonstrated by positive transillumination, positive radiographic studies, and antral puncture or displacement. Antral puncture and displacement are probably the most valuable criteria in diagnosing chronic suppurative sinusitis.

Treatment

The treatment for chronic suppurative sinusitis is primarily surgical. A small percentage of patients will get better using repeated irrigations or displacements, antihistamines, and antibiotics. In most patients, however, irreversible mucosal damage will make surgery necessary. The physician must first rule out any underlying illness such

as allergy hypothyroidism, decreased gamma globulin, anemia, or malnutrition, that may predispose the patient to infection. Surgery involves removal of all diseased soft tissue, postoperative drainage, and, if possible, obliteration of the sinus cavity. The Caldwell-Luc approach with inferior meatal nasoantral window is the most popular operation for chronic maxillary sinusitis.

ALLERGIC RHINITIS

Allergic rhinitis, an antigen-antibody hypersensitivity resulting in a perennially or seasonally stuffy nose, is accompanied by the excess production of clear mucus and some degree of anosmia. Because this is an allergic condition, it is important to obtain a thorough history in order to delineate the allergen. Allergic rhinitis may be classified as follows (Table 1). /3/

TABLE 1
CLASSIFICATION OF ALLERGIC RHINITIS

Seasonal	*Nonseasonal*
Spring – trees	Extrinsic
Summer – grasses	Inhalants
Fall – weeds	Incongestants
Insect Emanations	Contactants
Caddis fly	Infectants
Mayfly	Intrinsic
Fleas	Bacteria
Cockroaches	Viruses
Molds	
All year – indoors	
Spring to Fall – outdoors	

Signs and Symptoms

Allergic rhinitis produces symptoms similar to sinusitis but the mechanism is much different. It begins with deposit of an antigen on the nasal mucosa, the most common antigen being a pollen particle. Because of their minute size (25 to 40 μ), pollen particles tend to stay in the nose. Lyzozymes in the nasal mucosa dissolve the carbohydrate coat and the remaining protein is absorbed via the circulation; thus the antigen finds its way into the reticuloendothelial system. The presence of antigen in the cytoplasm of the plasma cells stimulates these cells to produce antibodies which are then released into the blood and attached to receptor cells similar to mast cells. When the antigen is again introduced, it unites with the previously formed antibody, causing a disruption of the mast cells and subsequent release of histamine, serotonin, heparin, and slow-reacting substance A. These chemicals produce the edema and smooth muscle spasm which are responsible for the symptoms of allergic disease.* The nasal mucosa can be a true shock organ.

The usual symptoms of allergic rhinitis are obstruction of the nose, frequent sneezing, and recurrent, thin nasal discharge as opposed to the thick discharge of sinusitis. /3/ Itching of the nose and eyes is common and frontal headache is not unusual. On physical examination, the nasal mucosa is typically edematous and pale, perhaps bluish, especially around the inferior turbinate. In children, look for "allergic shiners" under the eyes, Denny's lines, and a broadened nose secondary to the "allergic salute". /4/

To aid in the diagnosis, a nasal mucosa smear with cottontip applicator may be obtained and stained with Hansel stain. This will stain the red cytoplasmic granules in the eosinophils for indentification. In allergic rhinitis, the number of eosinophils may run as high as 90 percent. More than 3 to 4 percent of eosinophils in a nasal smear suggests that allergy may be the underlying cause of the patient's

---

* *Allergy for the Practicing Physician.* A collection of articles (unpublished), Methodist Hospital Graduate Medical Center, Indianapolis, Indiana.

nasal complaint. Radiographs of the paranasal sinuses are needed only to rule out sinusitis, and not for diagnosis, although they may show mucosal thickening.

Treatment

Treatment of allergic rhinitis is based on interrupting the pathophysiologic sequence. An attempt must first be made to eliminate the allergenic source. If the specific allergen is determined by specific sensitivity testing, hyposensitization can be initiated; this method is quite successful in treating hayfever, but not very helpful with molds or animal dander. A nasal obstruction can aggravate an allergic condition and should, therefore, be corrected. Therapy with antihistamines which block the histamine receptor sites and have an atropinic side effect to help dry nasal secretions is useful. The common side effect of drowsiness from antihistamines can frequently be countered by combining them with ephedrine (Sudafed®) or by switching to a different class of antihistamines. These drugs tend to work well in the early seasonal allergy but wane in effectiveness as the season progresses. Systemic sympathomimetics may be used to shrink the nasal mucosa and correct possible cholinergic overload producing the nasal discharge. Steroid sprays such as dexamethason (Turbinaire®) are quite effective in treating seasonal rhinitis. Response to treatment varies and is proportional to how well the allergen can be avoided and to what degree hyposensitization helps. Good results are possible with treatment; it is important to realize, however, that the allergy can be aggravated at any time, making the problem a chronic one. A few complications of allergic rhinitis are noteworthy: (1) Hypertrophied turbinates may lead to nasal obstruction from long-standing allergic rhinitis. (2) Nasal polyps are frequently seen in patients with allergic rhinitis. (3) Asthma and other allergic respiratory problems are sometimes present in patients with allergic rhinitis.

VASOMOTOR RHINITIS

Vasomotor rhinitis is produced by one or more factors which cause mucosal edema and a decrease or increase in nasal discharge with generally increased viscosity. The exact mechanism of vasomotor rhinitis is individual and not fully known. Vasomotor rhinitis may conveniently be classified by etiology (Table 2). /3/

TABLE 2
CLASSIFICATION OF VASOMOTOR RHINITIS

Neurogenic	*Chemical Agents*
Stress	Benzene
Anxiety	Tobacco smoke
Endocrine	*Chronic Nasal Irritation*
Pregnancy	Nose drops
Oral contraceptives	Nose picking
Hypothyroidism	*Occupational*
Mechanical	Dust
Nasal polyps	Extreme temperatures
Physical Agents	
Draft on patient's back	

Signs and Symptoms

The signs and symptoms of vasomotor rhinitis are quite similar to those of allergic rhinitis. Many patients complain of chronic, intermittent nasal stuffiness, accompanied by decreased or increased, often viscid, nasal discharge. The symptoms are common complaints of tense, nervous patients. Patients suffering from hypothyroidism, menopausal women, and patients on estrogen therapy are prone to complain of the signs and symptoms of vasomotor rhinitis. Expectant mothers near the end of the first trimester may develop nasal obstruction which returns to normal following delivery. A thorough physical examination which includes a nasal smear,

a thyroid workup, checking for a deviated septum, and querying the patient regarding pregnancy, is essential.

In some patients, signs and symptoms of allergic rhinitis (pruritis of the eyes and nose, lacrimation, and sneezing) are present but no history of allergy or infection can be elicited. Vasomotor rhinitis is usually responsible for these symptoms.

Treatment

The underlying cause, if identifiable, is important in the treatment of vasomotor rhinitis. For the nervous or tense patient, reassurance is often all that is necessary. Others may require tranquilizers or psychiatric evaluation. The hypothyroid patient may benefit from thyroid extract, the pregnant patient is usually cured by delivery, the patient with polyps or a deviated septum may be aided by surgery, and the patient affected by extremes of temperature may need a constant environment. After the underlying causes of vasomotor rhinitis have been treated, symptomatic therapy with a decongestant is about all that can be done. Increasing nasal moisture with normal saline, glyceryl, guiacolate, or potassium iodide also help.

The response to treatment of vasomotor rhinitis depends upon accuracy in recognizing the predisposing factors and on the emotional makeup of the patient. Those problems of a neuropsychiatric origin can be most difficult and exasperating for the physician.

CONCLUSION

As is true with most illnesses, those of the nose and paranasal sinus respond best to accurate diagnosis and appropriate and timely treatment.

References

1. Smith JM, Smith IM: The medical treatment of sinusitis. Otolaryngol Clin North Am 4:39-56, 1971.
2. Catlin FI, Cluff LE, Reynolds RC: The bacteriology of acute and chronic sinusitis. South Med J 58:1497-1502, 1965.
3. Taub SJ: Some aspects of nasal allergy: a review. Eye Ear Nose Throat Mon 53:158-159, 1974.
4. Stroh JE: Allergic rhinitis. Postgrad Med 45:151-154, 1969.

THE RED EYE

COL Floyd L. Wergeland, Jr., MC

An inflammation of the eye can be an uncomfortable situation for the patient and a real problem for the physician. The signs demonstrated on eye examination and the patient's complaints may help the physician decide whether to treat the condition or to refer the patient to an ophthalmologist for specialized care.

GENERAL EVALUATION

The patient's symptoms of particular interest to the physician are ocular or orbital pain, change in visual acuity, and the presence of a discharge. The signs of importance are clarity of the ocular media, size of the pupils, location and degree of ocular injection, and depth of the anterior chamber. If indicated by the findings, the intraocular pressure should be measured. A history of trauma will also aid in narrowing the differential diagnosis.

The eye examination is facilitated by several items of equipment. These include a good focal light, a loop magnifier, topical eye anesthetics, fluorescein dye strips, cotton applicator sticks, a foreign body spud or a hypodermic needle, and a tonometer. As an aid to differential diagnosis, the signs and symptoms of the conditions which produce a red eye are included in the Table.

TRAUMA AND RELATED CONDITIONS

Ocular Foreign Body

An occupational history of drilling or pounding is suggestive of this diagnosis. The eye may be "bloodshot"

TABLE
SIGNS AND SYMPTOMS
OF THE CONDITIONS WHICH PRODUCE THE RED EYE

Signs and Symptoms	Ocular Foreign Body	Corneal Abrasion	Subconjunctival hemorrhage	Corneal Burns	Acute Conjunctivitis	Episcleritis & Scleritis	Keratitis	Corneal Ulcers	Acute Closed Angle Glaucoma	Acute Iritis	Posterior Uveitis
Pain	Moderate to severe	Moderate to severe	Minimal or none	Minimal to moderate	Minimal or none	Tenderness	Minimal to moderate	Minimal to moderate	Usually severe	Moderate to severe	Minimal
Vision	Variable	Variable	Normal	Decreased	Normal	Normal	Decreased	Usually decreased	Usually markedly decreased	Slightly to markedly decreased	Decreased
Discharge	Watery	Watery	None	Watery	Moderate to copious	None	Watery	Moderate	None	None	None
Media	Normal	Irregular corneal light reflex	Clear	Cornea hazy	Cornea clear	Clear to hazy cornea	Hazy	Local haziness	Steamy cornea	Cornea clear to hazy	Hazy vitreous
Pupil	Normal	Normal	Normal	May be smaller	Normal	Normal	May be smaller	May be smaller	Dilated & fixed	Smaller & sluggish	Normal
Injection	None to mild	Diffuse	None	Diffuse or local	Diffuse, located near fornices	Quadratic	Perilimbal	Localized limbal or diffuse perilimbal	Perilimbal	Perilimbal	Mild & diffuse
Anterior Chamber	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Shallow	Normal	Normal
Iris	Normal	Normal	Normal	Normal	Normal	Normal or muddy	May be muddy	May be muddy	Mildly muddy marking	Muddy marking	Normal
Intraocular Pressure	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Elevated	Usually normal	Normal

or diffusely injected, tearing, and the patient may complain of moderate pain. If a foreign body cannot be seen on the cornea, one should evert the upper eyelid with a cotton applicator. If the foreign body is located, anesthetize the cornea with topical tetracaine and remove the object with a swab. A foreign body embedded on the cornea is usually visible when about 1 mm in size. However, a drop of 1 to 2 percent fluorescein dye can highlight the foreign body and demonstrate possible areas of denuded corneal epithelium. A corneal foreign body can frequently be removed from the anesthetized cornea by irrigation with a saline solution. If this is not successful, one can slowly dissect the foreign body from the cornea with a sterile hypodermic needle, keeping the needle parallel to the surface of the cornea. No attempt should be made to wipe off the particle because this usually results in further abrasion of the cornea. Therapy consists of applying an antibiotic ointment such as Neosporin,® patching the eye firmly, and examining the patient daily until the corneal epithelium is replaced.

Corneal Abrasion

A corneal abrasion may present with symptoms similar to those of a foreign body. Corneal abrasion is frequently caused by injury from an item such as a stick, fingernail, the edge of a sheet of paper, or a small missile. The injury produces marked tearing, spasms of the eyelids, diffuse ocular injection, pain, and a foreign body sensation. The instillation of a drop of fluorescein dye will outline the denuded corneal epithelium by appearing as a bright yellow-green stain of the cornea. One should always search for possible foreign bodies on the surface of the eye or under the lid. Treatment consists of instilling a topical anesthetic, 2 percent homatropine solution, and a broad spectrum antibiotic ointment, followed by application of a firm eye dressing for 12 to 24 hours. The ocular pain can be controlled with oral medication after the patch is applied. Do not give the patient topical anesthetic drops to use at home; its continuous use can interfere with the metabolism of corneal epithelium and eventually produce total loss of the corneal layer, thereby prolonging the healing process.

Subconjunctival Hemorrhage

A spontaneous subconjunctival hemorrhage causes a painless red eye. Such a hemorrhage may occur at any age, but is more prevalent in older people. The patient often does not realize that he has a "blood spot on the white of the eye" until his attention is directed to it. The hemorrhage seemingly occurs spontaneously but may be caused by rubbing the eye. It is usually localized in one quadrant. No specific treatment is required except to reassure the patient that the hemorrhage will clear in approximately two weeks. One should examine the anterior chamber and fundus for gross blood. If the hemorrhage recurs frequently, the patient should probably be evaluated for hypertension and blood dyscrasia.

Corneal Burns

Ultraviolet or infrared flash burns occur following overexposure to sunlamps or arc welding. If protective goggles are not worn, a superficial keratitis can be produced, usually within a few hours. The eyes become red and painful with increased photophobia. After instilling fluorescein dye the cornea will stain faintly and diffusely. Treatment consists of topical antibiotic ointment and a firm eye dressing. Analgesia is accomplished by oral medication.

Traumatic Iritis

Not infrequently, a contusion of the eye will produce a traumatic iritis. Visual acuity is moderately reduced and the eye pain is localized to the eyeball and supraorbital region. Conjunctival circumcorneal hyperemia fades toward the conjunctival cul-de-sac. The pupil is slightly constricted compared to the other eye and it reacts sluggishly to direct light. The patient usually

complains of photophobia. After a thorough examination for other trauma-related injuries, treatment consists of dilation of the pupil and frequent application of topical steroids.

EXTERNAL OCULAR INFLAMMATION

Conjunctivitis

Conjunctivitis is suspected when inflammation of the conjunctiva produces injection of the conjunctival vessels and redness of the bulbar conjunctiva. The hyperemia fades toward the limbus and moves with the conjunctiva; the margins of the lids may be edematous and injected. The patient with conjunctivitis experiences a sense of discomfort or burning; if an allergy exists, there may also be marked itching. The lids of the inflamed eye may stick together in the morning with either a purulent or mucopurulent discharge. If discharge is abundant, as is common in bacterial conjunctivitis, the vision may be slightly decreased. In viral infections the discharge is watery and the preauricular lymph nodes on the involved side may be enlarged and tender.

Treatment consists of a conjunctival culture to determine the specific causative agent, followed by use of a broad spectrum ophthalmic antibiotic and warm, moist compresses. If no improvement is noted in three days, the patient should be referred to an ophthalmologist for more detailed examination and definitive therapy.

Episcleritis

In episcleritis there is usually a unilateral localized area of redness on occasion involving the entire eye. The injected episcleral vessels are red or faintly purplish in appearance. In contrast to a conjunctivitis, the inflamed area is tender to the touch through the overlying lid. A fairly common condition whose etiology is not

known, episcleritis is often present in the patient with an allergic background. It has been seen in association with rheumatoid arthritis, keratoconjunctivitis sicca, and coccidiodomycosis. Its localized nature, frequently on the temporal side of the globe, its absence of discharge, and its coexistence with a mild iritis separate it from a conjunctivitis. In most cases, the inflammation produces no complications and responds readily to topical steroid drops. In rare cases a small inflammatory nodule in episcleral tissue is firmly attached to the sclera; this nodular form is less responsive to therapy and may last many weeks to several months. Fortunately, both forms have a low incidence of recurrence.

Scleritis

Scleritis is a serious inflammatory process. Although it rarely occurs, once present, it may produce considerable pain and photophobia. The injected site may be diffuse, localized, nodular, or actually show ulceration. It may be associated with a severe anterior uveitis, arthritis, and systemic collagen disease. In the nodular type, there is an elevated, dark-blue area in the anterior portion of the sclera. The adjacent cornea may also be inflamed.

Unfortunately, scleritis is resistant to almost all types of therapy. Systemic and topical corticosteroids are of some benefit.

Keratitis

Inflammation of the cornea or keratitis is always a serious situation because improper management can result in permanent visual impairment, varying from slight blurring to total blindness. The eye is painful, with ciliary or conjunctival injection, or both. The corneal reflex may be irregular and rough in appearance. This corneal change causes decreased vision, especially when it is centrally located. There is no discharge except in the case of a purulent bacterial ulcer.

Of great importance is the recognition of the possible existence of herpes simplex (dendritic) keratitis. This virus produces corneal ulceration, almost always unilateral. It may affect any age group. The patient usually complains of mild irritation and photophobia. If the central portion of the cornea is involved, vision may be blurred. A history of a recent upper respiratory infection with cold sores of the face is commonly elicited.

Staining the cornea with fluorescein dye will highlight the corneal dendritic figure produced by ruptured vesicles of the epithelium. Another diagnostic aid is the presence of a hypesthetic to anesthetic cornea. The degree of anesthesia can be determined by comparing the cornea sensitivity of both eyes with a wisp of cotton from an applicator stick.

Progression of the disease may involve the corneal stroma with a hypopyon, perforation, and corneal vascularization with permanent scarring. Progression, fortunately, rarely occurs unless the initial epithelial ulcer is not properly managed. The therapeutic objective is to denude the virus-containing epithelium without disturbing the deeper layers of the cornea. This can be accomplished by mechanical removal of the infected area with a cotton swab. Also, antiviral agents such as idoxuridine applied topically can effectively treat the acute case.

Corticosteroids are contraindicated in all stages of this condition because they may increase the activity of the virus by immunosuppression and by enhancing the action of collagenase from the necrotic epithelium of the corneal stroma. All keratitades are true ophthalmic emergencies and are best treated by specialists.

Corneal Ulcers

In general, corneal ulcers are caused by a wide variety of conditions such as microorganisms (bacteria, viruses, and fungi), hypersensitivity reactions, avitaminosis, fifth nerve lesions, and exposure.

Corneal ulcers are seen as localized staining infiltrated areas on corneas with an associated conjunctivitis or iritis. There may be pus in the anterior chamber. The ulcers frequently follow trauma; those located near the corneal limbus are more commonly seen in patients with blepharitis or conjunctivitis. Once present, the ulcers require identification of the offending organism by culture. While waiting for the culture report, the patient should be treated with a broad spectrum antibiotic drop and referred to a specialist.

INTRAOCULAR INFLAMMATION

Acute Closed Angle Glaucoma

Acute closed angle glaucoma is a relatively rare type of glaucoma, but when full-blown is one of the most painful conditions known to man. The onset is sudden, with rapid loss of vision and severe ocular and periorbital pain. The eye becomes almost rock-hard. The greater and more sudden the rise of intraocular pressure the more intense the visual loss. The lids may be swollen and there may be circumcorneal and conjunctival injection and edema. The cornea shows marked decrease in transparency and the iris is indistinct because of corneal edema. The pupil is semidilated and fixed and the anterior chamber appears shallow. The patient may complain of severe headaches, associated nausea and vomiting, and seeing haloes around lights. Acute closed angle glaucoma most commonly occurs in the elderly farsighted individual, and can be precipitated with dilation of the pupil.

In some cases of acute and chronic iritis, glaucoma is superimposed because of the marked congestion and the presence of iris adhesions and cellular debris in the anterior chamber. The pain in these cases is not as severe nor does the condition result in as much visual loss as does an acute closed angle glaucoma.

Acute Iritis

It is always important to make the correct distinction between acute iritis and acute glaucoma because the treatment of the conditions is diametrically opposed. Unless one is sure of the diagnosis, no medications should be instituted.

Acute glaucoma demands intensive and immediate miotic therapy plus the use of oral carbonic anhydrase inhibitors or hyperosmotic agents. Acute iritis or iridocyclitis is managed by the use of topical cycloplegics and corticosteroids. Although on gross examination the two entities may be similar, closer examination will usually resolve the difference. The intraocular inflammation of iritis produces perilimbal injection, moderate pain, some decrease in vision, and occasional mild corneal edema, and white dot-like deposits on the posterior surface of the cornea. Of special importance is the fact that the pupil is frequently smaller than normal and sluggish to light. In addition, the anterior chamber is normal in depth and the intraocular pressure is usually normal. The ophthalmologist should definitely be consulted for treatment of this condition.

Posterior Uveitis

Chorioretinal inflammatory lesions of the posterior aspect of the eye, though not as severe in intensity, may produce the external clinical appearance of an acute iritis.

Posterior uveitis is the term used to identify either choroiditis or chorioretinitis since in most cases both structures are affected. The specific etiology of posterior uveitis may be difficult to determine, but evaluation for toxoplasmosis, tuberculosis, syphilis, sarcoidosis, and sympathetic ophthalmia are the prime areas of concern. The inflammatory process may involve the macular region of the retina, cause an extensive vitreous reaction, and

markedly reduce vision. The spillover of this reaction into the anterior segment of the eye presents an iritis-like picture. It is therefore important to complete the ocular examination with funduscopy. Again, this is a serious situation and warrants immediate therapy by an ophthalmologist.

CONCLUSION

Eye inflammations which produce a red eye are common medical problems and have many causes. They are most serious when vision is threatened or the intraocular pressure is elevated. A good working knowledge of the differential diagnosis is essential in order to insure proper management and referral. One should keep in mind several firm guidelines regarding treatment: (a) Do not prescribe steroid preparations alone or with antibiotics except for acute allergic conjunctivitis. (b) Do not instill atropine, for it may blur vision for as long as two weeks and may precipitate an attack of closed angle glaucoma. (c) Do not prescribe anesthetic drops or ointment as a form of treatment following examination of the eye or removal of a foreign body.

In conclusion, whenever any doubt in diagnosis exists, the patient should be referred immediately to an ophthalmologist for guidance and treatment.

MANAGEMENT OF VAGINAL DISCHARGE

COL Alfred L. Franger, MC

Vaginal discharge or leukorrhea is one of the most common semiemergency problems which the physician is called upon to manage. Fortunately, most cases of leukorrhea are simple to treat. On occasion, however, relieving the symtoms may be as frustrating to the doctor as it is for the patient. In fact, the persistent or recurrent discharge may be as taxing to the physician's diagnostic and therapeutic skills as is an unexplained anemia or cardiac arrhythmia.

Some of us tend to minimize the patient's complaints because the problem is not life-threatening. However, many women not only are extremely uncomfortable, but may also be concerned about the possibility of underlying disease such as venereal disease or cancer. (If the males among you can imagine a good case of "jock itch" with the addition of a continually wet bottom and a persistent disagreeable odor, you may begin to get a clue as to how bothersome the problem may be!) Thus, leukorrhea has many implications not only for the patient herself, but for her sexual partner, and possibly those around her, as well.

HISTORY

As with the management of any medical problem, a thorough history is the keystone to therapeutic success. Simply inquiring into all the details of quality, quantity, and chronology may seem to be overly time-consuming. Nevertheless, in the cases of resistant vaginitis this approach may give etiologic clues which the laboratory cannot begin to furnish. Not the least important part of the history is

a careful inquiry into the patient's social and sexual life. These details will often furnish useful insight into how the patient feels about herself and how she interacts with her environment. Any type of stress may play an important role in the genesis and maintenance of a vaginal discharge. Unresolved emotional conflict mediated through subcortical centers, the limbic system, or the visceral brain, may eventually be transmuted into systemic changes and psychosomatic disorders. Physiologic and psychologic stresses may alter the vaginal pH, the integrity of the vaginal cells, and the glycogen content of the vagina. Under these circumstances the protective effect of the normal vaginal flora is reduced and overgrowth of pathogens may occur. The inquiry into these extragenital historical factors should be made at the first visit and not postponed for several reasons. First, it may save the patient valuable time and money by early identification and management of the underlying psychosocial problems. Second, to postpone inquiry into psychosocial events until "organic disease" is ruled out may serve to fixate in the patient's mind the thought that "it's all in my head," an attitude not conducive to further therapy. Third, elucidation of physical and psychologic contributants simultaneously may enable the patient herself to make the connection between life events and recurrence of the vaginitis, an insight which is usually therapeutically helpful.

Not all complaints of leukorrhea are specifically due to vaginitis; the discharge may be secondary to other disorders. If accompanied by menstrual complaints, the discharge may be symptomatic of organic pelvic disease. A long-standing discharge may indicate cervical changes or endometrial polyps. Accompanying bouts of cystitis may indicate a primary urinary tract problem. In these instances the thorough history may be illuminating.

Other important historical points include the following:

- Long-term drug therapy which destroys the normal Döderlein's bacillus flora and predisposes to *Candida* infections.

- Nocturnal or early morning exacerbation of symptoms may indicate pinworm infection.
- Oral contraceptive usage may increase vaginal glycogen content and predispose to candidiasis.
- Genital problems in the sexual partner(s) may indicate the "ping pong" syndrome or passage of pathogens back and forth between the partners.
- Confining undergarments, particularly those made of tight, woven synthetic fibers, may cause an accumulation of perspiration in the vaginal-perineal area and predispose to secondary infection.
- Vaginal and perineal powders, sprays, perfumes, douches, and soaps may produce irritation and allergic reactions.
- Improper perineal hygiene (wiping the anus back-to-front) may cause fecal contamination of the vagina.
- Frequent douches may alter normal vaginal flora or the chemicals used in the douche may cause tissue damage.

PHYSICAL CHARACTERISTICS

Physical characteristics of leukorrhea accompanying specific infections are described in the Table. In other instances, the characteristics of leukorrhea may point to a noninfectious etiology. Purulent, mucoid, or blood-streaked discharge may indicate cervical or upper tract pathology including venereal infection. Thick, viscid, foul-smelling discharge may indicate an intravaginal

foreign body such as a forgotten tampon. Clear and mucoid or watery discharge may accompany certain high estrogen oral contraceptive usage. The leukorrhea associated with fallopian tube carcinoma is characteristically described as thin, copious, and straw-colored. Some women may also complain of a discharge which upon close questioning may be compatible with the vaginal lubrication present upon sexual excitement. This lubrication may occur even when the woman is not consciously aware of being sexually excited.

LABORATORY DIAGNOSIS

Laboratory diagnosis of the specific infectious agent is usually simple and is accomplished by office examination of a "wet prep" or hanging drop slide. To facilitate identification of causative organisms, the patient should be asked not to douche or have intercourse during the 24 to 48 hour period prior to her office visit. Contamination of the specimen should be avoided by the use of a dry, warm speculum. If a lubricant is necessary, warm water may be used. With a sterile swab a sample of the discharge is taken from either the posterior fornix or the lateral wall of the vagina. Two slides are prepared: one with normal saline and one with 10 percent potassium hydroxide (KOH) which will lyse the cellular elements and facilitate the visualization of *Candida* mycelia, if present. A cover slip is placed over the solution and the slides are immediately examined under a microscope. Staining is usually not necessary. This simple technique enables the physician to rapidly ascertain the etiology of most vaginal infections. If necessary, the vaginal and cervical swabs may be transferred to the appropriate medium for culture or stained for further identification.

CHARACTERISTICS OF NORMAL VAGINAL DISCHARGE

Before describing the specific vaginal infections, let us consider the physical, chemical, and microbiologic characteristics of the normal vaginal discharge. The normal discharge is a mixture of excretions, secretions, transudates, microorganisms, cellular debris, and various other ingredients. The cells present are largely those exfoliated from the vaginal walls although some may also come from the cervix and endometrium, especially near the onset of menses. In the early follicular phase the fluid is viscous and milky; it becomes more liquid and increases in quantity as the menstrual cycle progresses. During the brief ovulatory phase, the liquid increases in quantity and becomes more mucoid. Leukocytes are abundant and red blood cells may occasionally be noted. In the luteal phase, the secretions become thicker, the amount of mucus decreases, epithelial cells are predominant, and the number of bacteria increases. During the last two premenstrual days, the fluid may be milky with some increase in amount. Red and white blood cells are increased, squamous epithelial cells are abundant, and the bacteria show great variation in species.

The vaginal flora of women in their reproductive years is generally stable. Lactobacilli reappear at puberty and predominate until menopause. The Doderlein's bacillus is associated with low vaginal pH and total count increases during pregnancy. Three genera of yeast (*Candida*, *Cryptococcus*, and *Saccharomyces*) and a protozoan (*Trichomonas*) are also normal inhabitants. Chemically, many proteins and carbohydrates are present. The pH of the healthy vagina varies between 4 and 5, with higher pH present near the introitus and in the posterior fornix. The discharge associated with sexual excitement is a thin, slippery, cloudy transudate which becomes more alkaline with increased stimulation.

SPECIFIC VAGINAL INFECTIONS

The most frequent etiologic agents of vaginitis are the *Candida* species (of which there are several), *Trichomonas vaginalis*, and *Haemophilus vaginalis*. These three organisms are responsible for approximately 90 percent of all cases of vaginitis, and may exist singly or in combination. Less commonly involved organisms are streptococci, staphylococci, Escherichia coli, fusospirochetes, and the tubercle bacillus. These infrequently-appearing organisms usually exist as complications of other factors such as hypoestrinism, systemic illness, or medications, and often require no specific treatment other than correction of the primary etiologic factor. Last but not least, there is the sometimes seemingly prevalent so-called "nonspecific" vaginitis for which no etiologic agent can be found.

A synopsis of the characteristics of the "Big Three" causes of leukorrhea is found in the Table. Other important points to consider are as follows:

Candidiasis. Candida species organisms are indigenous to practically all humans and to many animals. For this reason the host factors controlling susceptibility are more significant in the development of an active infection than chance contamination of the vagina by a species of *Candida*. It is thus important to consider and rule out all predisposing factors. The amount of vulvar pruritus present is generally proportional to the vulvar erythema. It is important that a full course of prescribed local medication be used by the patient and continued through the menses, if necessary. This point should be stressed to the patient at the time of prescribing.

Trichomonas vaginalis. In an unfavorable environment (such as on a heated slide) the organism may be ball-shaped and difficult to distinguish from other cellular elements. *Trichomonas* vaginitis may also be considered as a venereal

TABLE
PHYSICAL CHARACTERISTICS OF LEUKORRHEA
ACCOMPANYING SPECIFIC INFECTIONS

Physical Characteristics	*Trichomonas Vaginalis**	*Candida* Species†	*Haemophilus Vaginalis*‡
Predisposing factors	Hyperestrinism; vaginal hypoacidity; lactobacillus decrease; stress	Increased vaginal glycogen content (pregnancy, oral contraceptives, systemic steroids, diabetes); systemic antibiotics	Estrogens
Complaints	Malodorous discharge; intolerable pruritus; chafing; urethrocystitis	Vulvar pruritus; vaginal itching; dysuria; dyspareunia	Malodorous discharge (mild)
Signs	Papular lesions in vagina ("strawberry vagina") and on cervix, possibly some vestibular erythema, intertrigo	Vulvar erythema (especially between labia; edema of labia minora; traumatic excoriations of vulva	Minimal to absent
Discharge	Profuse; frothy; green; foul-smelling; watery; may be yellow or gray; pH less than 5	Thick; white to yellow-white; "cottage-cheese"	Resembles thin flour paste; tends to adhere to vaginal wall; mostly gray, occasionally white or green; "disagreeable odor"
Wet prep findings	Actively motile flagellated organisms; may be only round, immature forms in asymptomatic patients	Branching pseudohyphae; mycelia (potassium hydroxide preparation)	Many stippled epithelial cells; few pus cells; lactobacilli absent
Special diagnostic procedures	Giemsa or acridine orange stain	Culture on Nickerson's medium	Gram stain - gram negative; small bacilli
Recurrent cases	May be reinfection from urethra or Skene's ducts	Mostly autogenous reinfection from clitoris or gastrointestinal tract	Probably reinfection from non-treated partner
Male factors	High possibility of asymptomatic lower genital tract infection; may have dysuria, discharge or postcoital burning	Organisms may be beneath foreskin or in semen	Organisms found in 90 percent of partners of infected women
Treatment - specific	Metronidazole, 0.25 gm three times a day for 10 days or 0.5 gm every 12 hours for 5 days	Local preparations (many)	Oxytetracycline vaginal suppositories at bedtime for 10 days or ampicillin 0.5 gm orally every 6 hours for 5 days
Treatment - adjunctive	Treat partner concurrently; acid douche; relieve stress	Oral medications for gastrointestinal tract; control other factors; condom for partner; steroids for vulvitis	Immediate postcoital douche or condom for partner

*Fungus
†Flagellated protozoan
‡Gram-negative bacillus

disease, and infection without intromission is possible. Inanimate objects such as douche tips, washcloths, and bath towels may also be sources of infection, but there is minimal chance of infection from bath tubs and toilet seats. This form of vaginitis tends to be chronic with acute exacerbations; the vagina of a patient with a chronic infection may show no changes. Diagnosis of *Trichomonas* vaginitis by pap smear is subject to a high degree of error, both positive and negative.

Haemophilus vaginalis. Perhaps about 90 percent of all so-called "nonspecific" vaginitis is caused by *Haemophilus vaginalis*. The bacteria are usually transmitted during intercourse and there is a high rate of reinfection from nontreated partners. About 25 percent of patients with *Trichomonas* vaginitis have an associated *Haemophilus vaginalis* infection. Signs and symptoms may easily be missed, and the patient may complain only of a need for frequent douching for cleanliness. The characteristic disagreeable odor of *Haemophilus vaginalis* is said to be the first clue to the presence of an infection. Three important points may be made concerning therapy in general:

- It is important to treat the sexual partner(s) concomitantly.

- Treatment of vaginitis with systemic agents may decrease the normal protective vaginal flora and permit an overgrowth of *Candida*. For this reason, it may be advisable to simultaneously prescribe a local antifungal agent.

- The incidence of vaginal discharges and infections of all types is increased during pregnancy, largely because of concomitant hormonal and metabolic changes. Although it may be impossible to eradicate the causative organism, the symptoms may generally be controlled by long-term administration of topical agents. Usually the symptoms will disappear spontaneously in the postpartum period. Asymptomatic infections during pregnancy as a rule are not treated.

NONSPECIFIC VAGINITIS

The definition of "nonspecific" vaginitis varies somewhat, but is usually taken to mean an unusual discharge not explained by a readily identifiable agent. It has traditionally been ascribed to a "mixed bacterial infection" but isolation of a predominant organism does not necessarily prove a relationship of that agent to the condition present. Some of the "nonspecific" cases may be induced by allergic states, altered vaginal physiology, or chemical irritants. Others may be the result of organic lesions such as cervicitis or changes in sexual performance pattern. In addition, an overly-fastidious woman may interpret the normal physiologic secretions as being indicative of some other problem. It is in this category of "nonspecific" vaginitis that a meticulous history may yield diagnostic and therapeutic dividends.

VENEREAL DISEASE

While a discussion of veneral disease is outside the scope of this paper, it deserves some consideration because of its prevalence. Gonorrhea is the most common venereal disease to present with a vaginal discharge, and may usually be diagnosed by the presence of a purulent discharge from the cervix accompanied by signs and symptoms of an acute pelvic or systemic infection. If any possibility exists of the presence of *Neisseria gonorrhoeae*, endocervical and urethral specimens should be taken for gram stain or inoculation onto Transgrow or Thayer-Martin™ medium. The remainder of the venereal diseases are diagnosed by their characteristic vulvar and vaginal lesions. It is also possible for a venereal disease and a vaginitis caused by the common organisms to coexist.

Similarly, antibiotic treatment of a venereal disease may alter the vaginal flora and permit overgrowth of pathogens. In any event, simultaneous systemic and local therrapy may be necessary to afford complete relief to the patient.

VAGINITIS IN THE NONREPRODUCTIVE YEARS

Vaginitis occurring at the extremes of reproductive life may present special problems because of the thinness of the unestrogenized vaginal mucosa and its easy susceptibility to trauma. In children, candidiasis is responsible for a large percentage of vulvovaginitis which usually responds to careful perineal hygiene. Vaginitis secondary to foreign bodies (hairpins, crayons, and other objects) is fairly common; thus a vaginal or rectal examination to rule out this possibility is mandatory. Pinworms in the pediatric patient should also be considered, and the child's mother instructed to take appropriate specimens. Less commonly, vaginitis may be secondary to systemic illnesses and childhood viral eruptions may involve the vulva and vagina. In young girls an acute inflammatory vulvovaginitis may be the first sign of a gonococcal infection. It is now recognized that children can contract a gonococcal infection from contact with warm, moist discharges on bedding.

In the postmenopausal woman, the so-called "atrophic" vaginitis may be a source of discomfort, soreness, and burning pain in the vulvar area. This entity is not a true vaginitis, however; the symptoms are usually caused by a thinned vaginal mucosa secondary to estrogen deficiency rather than a true infectious agent, and a secondary bacterial infection may actually be present. The condition can be diagnosed by simple inspection and visualization of a thin, pale mucosa with loss of the normal rugae. If doubt exists concerning the patient's hormonal status, a maturation index will reveal a predominance of parabasal cells with loss of the superficial squamous cells.

Estrogen replacement therapy will usually reverse the atrophic changes. With the recent controversy surrounding estrogen therapy and uterine cancer, local estrogen creams may be preferable to systemic medication because systemic absorption of the estrogen from the cream is minimal. When the vaginal mucosa is restored to its premenopausal condition any secondary invaders present will usually disappear spontaneously. If symptoms persist after the atrophic condition is corrected the patient should be reexamined for other pathogens and appropriate therapy should be instituted.

SUMMARY

In summary, vaginitis and its attendant annoying symptoms are common complaints in today's medical practice. Many organisms as well as other factors may be responsible. The vast majority of vaginitis is due to three organisms existing singly or in combination. Therapy is usually fairly straightforward and relief of the problem is possible in most instances. A thorough patient history is necessary to illuminate and minimize contributory factors. The importance of a full course of therapy should be stressed to the patient. Serious consideration should also be given to concurrent treatment of the sexual partner(s). If all else fails, refer the patient to your friendly gynecologist.

Suggested Reading

1. Gardner H, Kaufman P: Benign Diseases of the Vulva and Vagina. St. Louis, C.V. Mosby Co., 1969, pp. 149-297.
2. Friberg-Donohue H (ed): Vaginitis...An update. Patient Care 8:44-77, 1974.
3. Jorgensen V: Holistic therapy for nonpathogenic vaginitis. Contemp Obstet Gynecol 7:45-46, 1976.
4. Jeffcoate N: Principles of Gynecology, 4th ed. Boston, Butterworths, 1975, pp. 555-557.

The power of making a correct diagnosis is the key to all success in the treatment of skin diseases; without this faculty, the physician can never be a thorough dermatologist, and therapeutics at once cease to hold their proper position, and become empirical.

Louis A. Duhring (1845 to 1913)
American Journal of Syphilography and Dermatology. **2:104, 1871.**

COMMON DERMATOLOGICAL PROBLEMS

LTC Richard B. Odom, MC

This communication outlines the ten most common dermatological conditions seen at Brooke, Fitzsimons, Letterman, and Walter Reed Army Medical Centers and includes a brief summary on the clinical, pathogenetic and therapeutic aspects of those diseases. In 1972, Letterman Army Institute of Research began collecting data on dermatological problems from the four medical centers in an attempt to determine the type and frequency of diseases seen, to facilitate the systematic study of various dermatological conditions, and to provide a repository of information pertaining to dermatological diseases. Diagnoses listed in Table below represent the visits for only the top ten dermatological conditions during 1972; the total number of dermatology visits to the four medical centers approaches 85,000 annually.

TABLE

THE TEN MOST COMMON DERMATOLOGICAL CONDITIONS SEEN AT FOUR ARMY MEDICAL CENTERS IN 1972*

Rank	Dermatological condition	Visits	Percentage
1	Acne vulgaris	11,441	19.8
2	Verruca vulgaris	5,297	9.2
3	Actinic keratosis	4,130	7.1
4	Seborrheic dermatitis	2,993	5.2
5	Seborrheic keratosis	2,684	4.6
6	Basal cell carcinoma	2,463	4.3
7	Allergic contact dermatitis	2,410	4.2
8	Nevus, dermal	2,124	3.7
9	Atopic dermatitis	1,725	3.0
10	Psoriasis	1,540	2.7

*These dermatological conditions were seen at Brooke, Fitzsimons, Letterman, and Walter Reed Army Medical Centers.

ACNE VULGARIS

Acne

Acne is a chronic inflammatory disorder of the pilosebaceous follicles involving the face, chest, back, and upper arms. Lesions occur in areas of well-developed, active, sebaceous glands. Androgens play an essential role. Corynebacterium acnes are consistently cultured and probably involved in the pathogenesis of inflammatory lesions. Emotional stress, climate, and diet may be contributory factors in certain cases. The primary lesion is the comedone which may progress to an inflammatory papule, pustule, or cyst. Superficial pits or deep, irregular scars may result from persistent inflammatory lesions. Disease activity usually diminishes with age. The objective of treatment is to reduce the severity of lesions which will lower the risk of subsequent scarring. Topical therapy includes various soaps, keratolytics, drying agents, benzoyl peroxide, and retinoic acid. Tetracycline or other antibiotics are necessary for inflammatory lesions. Topical antibiotics (tetracycline, erythromycin, and cleocin) are currently receiving wide use by dermatologists with promising results. Acne surgery and intralesional steroids are other useful modalities.

VERRUCA VULGARIS

Verrucae

Warts are caused by a group of DNA viruses (papovaviruses). The incidence of warts increases during childhood and peaks at adolescence. The mode of transmission is by direct contact and autoinoculation occurs. Histopathology is characteristic. Common warts (verruca vulgaris) are firm, asymptomatic, rough papules that may occur anywhere, but are most common on the hands and knees.

Flat warts (verruca plana) are smooth, skin-colored, slightly elevated papules commonly seen on the face, dorsum of the hands, and lower legs. Filiform (digitate) warts occur on the face, scalp, and neck. Plantar warts are well-demarcated, often painful keratotic papules commonly occurring beneath pressure points. Scalpel paring of the wart demonstrates small bleeding points that help to differentiate it from a callosity or corn. Venereal warts (condyloma accuminata) are flesh-colored, most cauliflower masses occurring in the genital area. Many warts resolve spontaneously over a period of months. Destructive measures--topical application of strong acids, 40% salicyclic acid plaster, electrodesiccation and curettage, carbon dioxide snow, cantharidin and liquid nitrogen--usually provide satisfactory treatment. Immunotherapy utilizing dinitrochlorobenzene, trichophytin, and rhus oleoresin as the eliciting antigens are employed successfully in some refractory cases. Venereal warts are best treated with podophyllin.

ACTINIC KERATOSIS

The most common precancerous skin lesion is the actinic keratosis (senile keratosis) usually appearing after the fourth decade in predisposed persons with excessive solar exposure. It is frequently associated with freckled pigmentation and yellowed, wrinkled skin due to ultraviolet damage (actinic elastosis). The keratosis may vary from skin-colored to brownish-black in color depending on the degree of adherent horny material. The keratinous surface is usually firmly attached to the underlying skin; it may be extremely verrucous and present as a cutaneous horn. Susceptible persons usually have multiple lesions over the sun-exposed areas: face, ears, scalp (particularly if alopecic), dorsum of the hands, arms, and legs.

Liquid nitrogen is a superior method of treatment in patients who have a small number of lesions. In those patients with numerous lesions of varying sizes,

1% 5-fluorouracil (5-FU) applied topically is the treatment of choice; 5% 5-FU may be required for treating the scalp, dorsum of the hands, and arms. Without treatment, actinic keratoses may invade the dermis as squamous cell carcinoma, but metastasis rarely, if ever, occurs.

SEBORRHEIC DERMATITIS

Seborrheic dermatitis usually starts in childhood and may continue throughout life. It is a common, mildly pruritic, inflammatory condition characterized by greasy, pinkish-yellow scales involving the scalp (cradle cap in infants), eyebrows, nasolabial folds, blepharal margins, ears, sternal area, axillae, and groin. Although the cause of this dermatitis remains unknown, active sebaceous glands are necessary for seborrheic dermatitis to develop.

Exaggerated forms of seborrhea and seborrheic dermatitis may appear in patients with parkinsonism or with brain disease. Elderly, debilitated, or obese adults with severe intertriginous involvement are most prone to the development of the rare exfoliative erythroderma of seborrheic dermatitis. Leiner's disease consists of generalized seborrheic dermatitis in the neonate, severe diarrhea, recurrent infections, and failure to thrive.

Seborrheic dermatitis of the scalp is fairly well controlled by medicated shampoos containing active ingredients such as sulfur, salicylic acid, resorcinol, tars, hexachlorophene, and selenium sulfide. Topical corticosteroids are the most active preparations in seborrheic dermatitis. Patients should be instructed that seborrheic dermatitis pursues a chronic course with remission and exacerbations.

SEBORRHEIC KERATOSIS

Seborrheic keratosis is a benign epidermal tumor of the skin varying from tiny, round, slightly elevated, flesh-colored or brown papules to large, round, or oval convex papules covered with greasy yellow scales. Occasionally the lesion may be black and simulate a malignant melanoma. Though more common in patients beyond the third and fourth decades, the lesions may also be seen in younger adults. Seborrheic keratosis is common in the upper part of the trunk, face, neck, axillae, groin, and extremities. The lesions are often multiple. Several cases have been reported of patients in whom the sudden appearance and rapid increase in size and number of freckles and seborrheic keratoses (sign of Trélat) were accompanied by manifestation and spreading of an internal cancer.

Although seborrheic keratosis is usually asymptomatic, many patients desire removal for cosmetic purposes. Liquid nitrogen is a superior method of eradicating these lesions.

BASAL CELL CARCINOMA

The predominance of basal cell cancer on exposed areas of the skin has long been recognized, and it is the assumption of many that actinic radiation is a major etiologic factor. Though basal cell carcinoma constitutes one of the least aggressive neoplasms among cancers, its capacity for local destruction underscores its malignant character. To date, less than 100 basal cell carcinomas have been known to metastasize.

The characteristic basal cell carcinoma is a small, pearly, smooth-surfaced nodule with telangiectatic vessels coursing over its rolled border. Occasionally the lesion may present as an area of atrophy or sclerosis or as an ulcer that fails to heal. The tumor enlarges quite slowly over a period of months and years.

Patients with light complexions have a propensity to develop basal cell carcinomas and signs of actinic damage. Few other concomitant lesions are noted. Exceptions to this general rule are found in those patients whose tumors are due to arsenic intake, where internal cancers may occur simultaneously with cutaneous tumors, and in patients with the nevoid basal cell carcinoma syndrome.

Many forms of therapy are effective in eliminating the basal cell carcinoma, and the choice of therapy must be individualized depending on several factors including age of the patient, prior radiation therapy, location of the lesion, and availability of the various modalities. Effective modalities include electrodesiccation and curettage, excision, cryosurgery, x-ray therapy, topical 5-FU, Moh's chemosurgery, and fresh tissue chemosurgery.

ALLERGIC CONTACT DERMATITIS

Allergic contact dermatitis (dermatitis venenata) results from exposure of sensitized individuals to contact allergens or sensitizers. It is a delayed hypersensitivity reaction in contrast to dermatoses that are of the immediate urticarial variety.

Acute dermatitis is characterized by redness, edema, papules, vesiculation, weeping and crusting, accompanied by pruritis. In the chronic phase, thickening, lichenification, fissuration, and pigmentation may occur. The clinical picture may be closely simulated by other eczematous dermatoses.

A vast number of allergens or sensitizers may cause allergic eczematous contact dermatitis. The six leading common causes in order of frequency are as follows:

- Rhus (poison ivy, oak or sumac)
- Paraphenylenediamine (hair dye)
- Nickel compounds
- Rubber compounds
- Ethylenediamine (stabilizer)
- Dichromates (cement and shoe dermatitis)

The patch test is indispensable in proving the cause of allergic contact dermatitis.

In all stages, topical therapy consisting of cool, wet dressings, and corticosteroid medications is used to alleviate itching and inflammation and to prevent scratching. In extensive cases or in cases with marked edema of the face, eyelids, or genitalia, systemic corticosteroids are indicated. Antihistamines may also be employed for their sedative and antipruritic effects. The only effective prophylaxis is to avoid contact with the offending allergen.

NEVI

Nevi are proliferative aggregates of apparently normal melanocytes. The common mole (pigmented nevus) may be flat, elevated, hairy, or pedunculated. It may even be nonpigmented. Moles are generally absent at birth, but may appear with increasing age, with changes in color, morphology, and size. During pregnancy, new moles may appear and previously existing ones may darken. Moles are found anywhere on the body surface, including the genitalia.

Common moles are generally described on the basis of three histologic patterns: junctional, compound, and dermal.

Malignant melanoma is extremely rare in children and most arise *de novo*, independent of preexisting nevi. Malignant melanoma should be suspected whenever a nevus rapidly enlarges, darkens, ulcerates, bleeds, displays a variety of color patterns, or becomes painful or pruritic.

Common moles are frequently removed for cosmetic purposes by shave or excision biopsy. If a pigmented lesion is suggestive of a malignant melanoma, a thorough excisional biopsy should be performed to allow assessment of the depth of invasion of the lesion.

Giant pigmented nevi that cover extensive areas of the body and significant congenital nevi should probably be removed prophylactically since there is a relatively high incidence of malignant transformation.

ATOPIC DERMATITIS

Atopic dermatitis is a common, chronic, markedly pruritic disorder of unknown etiology. The family history is frequently positive for hay fever, asthma, and atopic dermatitis. The infantile form is characterized by poorly demarcated, symmetrical, erythematous patches on the cheeks and extensor areas of the extremities. The chest, back, and diaper area may be involved. The onset, usually within the first year, is uncommon before the second month. Weeping, oozing, and crust formation usually occur. The skin markings on the palms are often accentuated. The childhood form is characterized by a dry, papular, lichenified dermatitis involving the antecubital and popliteal fossae, neck, and flexor surfaces of the wrists. Postinflammatory pigmentary changes and persistent wrinkling of the eyelids are often present. The adolescent or adult form is usually localized to the dorsal surfaces of the hands and feet and genitalia. The atopic skin, when irritated, becomes

unusually pruritic for a prolonged period of time. Factors that may exacerbate the disease include the extremes of heat and humidity, perspiration, rough clothing, fatigue, emotional stress, and infection. Most patients with active severe disease have elevated serum levels of IgE. White dermographism and the delayed blanch phenomenon with acetylcholine appear to be characteristic of atopic individuals. Complications include bacterial and viral skin infections (Kaposi's varicelliform eruption) with vaccinia virus (eczema vaccinatum) and herpes virus (eczema herpeticum). Differential diagnoses may include seborrheic dermatitis which occurs on the scalp (with greasy scales), diaper area, and axillae. Seborrheic dermatitis is less pruritic and usually responds rapidly to therapy including topical steroids, tars, compresses, soap substitutes, bath oils, antihistamines, and soft clothing. The patient usually improves with age but the disease often persists in a more localized form, into adulthood.

PSORIASIS

Psoriasis is a common, chronic, papulosquamous disease characterized by well-demarcated, erythematous patches that vary in size and are usually covered with a thick, silvery scale. The most common locations include the scalp, elbows, knees, gluteal cleft, lower back, nails and extremities.

Acute guttate psoriasis is a common childhood variant that may follow an upper respiratory or streptococcal infection. New lesions may develop in areas of trauma (Koebner's phenomenon). The nails may be orange-colored and demonstrate tiny pits, onycholysis or subungual keratosis. A positive family history is common. A seronegative, asymmetrical arthropathy may occur. The etiology of psoriasis is unknown, though the epidermal cell turnover time is markedly accelerated. Histopathology is usually diagnostic. The course is chronic and recurrent; patients often improve in summer. Differential diagnoses include

seborrheic dermatitis, pityriasis rosea, lichen planus, and secondary syphilis.

Most patients improve with conservative topical therapy using combinations of steroids, tars plus ultraviolet light (Goeckerman treatment), and shampoos. Currently oral psoralen and long-wave ultraviolet light therapy (PUVA) is being investigated under strict protocol. However, a number of important questions need to be answered and this form of chemotherapy cannot be recommended until these questions have been evaluated by controlled analytical procedures. Methotrexate should be reserved for individuals with extensive, recalcitrant psoriasis who are physically debilitated and socially crippled.

TESTING THYROID FUNCTION

LTC Harvey L. Eichner, MC

If all patients with Graves' disease had large, diffuse goiters, bulging eyes, obvious tremor, and perpetual activity, the diagnosis would be made in the waiting room. By the same token, patients with yellow, puffed faces, hoarseness, and constant sleepiness could be wheeled right past the laboratory en route to the pharmacy for thyroid hormone replacement. Unfortunately, the clinical suspicion of thyroid disease often rests on findings which are much more subtle and which require confirmation in the laboratory. The profusion of available thyroid function tests often creates more consternation than enlightenment, as their selection and interpretation become more complex (Table 1). The failure of any single test to provide conclusive evidence of disease is a major factor responsible for this complexity, but increased awareness of previously-unrecognized syndromes has also contributed to it.

TABLE 1

THYROID FUNCTION TESTS

- *Hypothalamic–Pituitary* Function
  - Thyrotrophin (TSH) radioimmunoassay
  - Thyrotrophin-releasing hormone (TRH) test
- Glandular Function
  - Thyroxine (T_4) assays
  - Triiodothyronine (T_3) assays
  - Thyroid hormone binding tests (thyroxine binding globulin (TBG) uptake, resin T_3 uptake)
  - Free thyroxine index (FTI) or effective thyroxine ratio (ETR)
  - Radioisotope uptake studies (^{131}I, ^{123}I, ^{99m}Tc)
  - TSH stimulation test
  - T_3 or T_4 suppression test
  - TRH test
- Peripheral Thyroid Effects
  - Photomotogram
  - Electrocardiogram
  - Basal metabolic rate
  - Serum cholesterol

Present Concepts, Ambulatory Internal Medicine Care Symposium, Summer 1977

History

The first tests of thyroid functional status were directed at the peripheral effects of altered thyroid secretion. Magnus-Levy /1/ in 1895 described the increase in oxygen consumption associated with administration of thyroid extract which formed the basis for the basal metabolic rate (BMR) measurement. In 1924, Chaney /2/ began the quantitative assessment of the Achilles tendon reflex in thyroid disease using a mechanical device which predated the more accurate photomotograph. The lack of specificity in this type of study created a desire for chemical measurement of thyroid secretion; however, these assays had to await identification of the thyroid hormones. It was initially believed that thyroglobulin was the circulating thyroid hormone because of its presence in extracts of thyroid glands. /3/ Despite the discovery of thyroxine (T_4) in 1915 by Kendall, /4/ it was not until 1940 that thyroglobulin was proved not to be the active hormone. /5/ And the second thyroid hormone, triiodothyronine (T_3), was not discovered until 1952. /6/ The first chemical assay of thyroid function, the protein-bound iodine, came into use in the 1950s, /7/ followed by thyroxine iodine measurement after column chromatography /8/ and followed finally by competitive protein binding (CPB) and radioimmunoassay (RIA) techniques /9/ in the past decade.

Normal Thyroid Gland

The normal thyroid gland, under the negative feedback control of pituitary thyrotrophin (TSH), concentrates iodine from the blood and then iodinates and couples tyrosine residues while they remain incorporated in thyroglobulin chains. After a period of storage, thyroglobulin is hydrolyzed by the thyroid cells to release T_4 and T_3 into the circulation where most are bound to an alpha-globulin, thyroxine binding globulin (TBG), and smaller amounts to pre-albumin and albumin. Although over 99.5 percent of the circulating T_4 and T_3 are so bound, the remaining free T_4 determines the thyroid functional status which is unaffected by changes in the binding proteins and their thyroid hormone passengers.

STANDARD THYROID TESTS

Measurement of TSH in the circulation would be valuable in primary hypothyroidism when TSH should be elevated; it would not be elevated in pituitary insufficiency. Because of lack of sensitivity of the assay at low serum levels, TSH decreases cannot be determined accurately and therefore TSH is not a part of the assessment for thyrotoxicosis. (Pituitary TSH hypersecretion is rare). The iodine-concentrating ability of the thyroid is measured by counting radioactivity over the thyroid gland following a dose of a radioisotope of iodine. While the radioiodine uptake is still useful in evaluating patients with thyrotoxicosis, increasing dietary and medical iodine intake have pushed the lower limits of normal radioiodine retention well into the old hypothyroid range, greatly restricting the utility of this test. Specific tests for functional defects in thyroid hormone synthesis are also available, but these are rarely used clinically.

The measurement of circulating thyroid hormone by any test other than CPB or RIA is no longer valid and there is no reason to discuss the protein-bound iodine, butanol-extractable iodine (BEI), or T_4 by column chromatography. However, because of the high proportion of bound versus unbound T_4 and T_3, any change in binding protein levels, especially that of TBG, will influence the test for total serum T_4 or T_3. Free thyroid hormone levels can be measured directly after difficult separation techniques, but the cost and error thereby introduced make such assays undesirable. An estimation of free T_4 can be obtained more easily with an index based on the total T_4 balanced by an inverse estimation of free TBG binding sites, the resin T_3 uptake test. This test is a measure of the radiolabeled T_3 remaining unbound to TBG, which is picked up by a resin sponge after a known amount is added to the patient's serum. It provides knowledge of the available binding sites for thyroid hormones on serum proteins. It is important to grasp the concept that resin T_3 uptake (RT_3U) increases in both hyperthyroidism

(protein constant, total T_4 increased) and in states of decreased binding protein (total T_4 decreased but not free T_4), because in both situations the available binding sites decrease, allowing more uptake by the resin sponge. Conversely, both hypothyroidism and increased binding protein cause a diminished RT_3U as the available binding sites on the patient's serum take up the radiolabeled T_3. Since changes in thyroid binding proteins push the T_4 assay and RT_3U in opposite directions, one can construct an equation which yields a free thyroxine index (FTI) to cancel out these changes. An elevated T_4 and RT_3U thus produce a high FTI indicating hyperthyroidism, while a high T_4 and low RT_3U secondary to increased binding proteins will give a normal FTI. The FTI based on T_4 by CPB or RIA, and the RT_3U are now the most important bits of information about thyroid function and the FTI is unaffected by iodine intake or abnormal serum protein concentrations (Table 2).

TABLE 2
CLINICAL STATES AND ALTERATIONS IN THYROID FUNCTION TESTS

	Thyroid Function Tests			
Clinical States	T_4*	RT_3U†	TBG (TBPA)‡	FTI§
Hyperthyroidism	H	H	L (L)	H
Hypothyroidism	L	L	H (H)	L
Pregnancy	H	L	H (L term)	N
Nephrosis	L	H	L	N
Liver disease	H, N, L	H, N, L	H, N, L	H, N, L
Protein deficiency	L	H	L	N
Hereditary TBG‡				
Excess	H	L	H (N)	N
Deficiency	L	H	L (N)	N
Drugs				
Dilantin	L	H	L (N)	N
High dose salicylates	L	H	N (L)	N
Estrogens	H	L	H (N)	N
Androgens, anabolic steroids, glucocorticoids	L	H	L (H)	N

*T_4 = Thyroxine
†RT_3U = Resin T_3 uptake
‡TBG (TBPA) = Thyroxine binding globulin (TBG). Values in parentheses are those of the second major thyroid hormone binding protein, thyroxine binding prealbumin (TBPA).
§FTI = Free thyroxine index
Symbols in the Table: H = High; N = Normal; L = Low

The T_3 assay, which should not be confused with the resin T_3 uptake, is a direct assay of serum T_3 level. It is also affected by changes in binding protein, and although a free T_3 index is not generally calculated, the RT_3U may be useful as an aid in interpreting abnormal T_3 levels. The measurement of serum T3 is indicated whenever hyperthyroidism is suspected since it invariably is elevated in thyrotoxicosis while the T_4 will occasionally be normal or only slightly elevated. However, since the thyroid will preferentially manufacture T_3 to conserve iodine in states of TSH elevation, the T_3 assay is not as valuable in the diagnosis of hypothyroidism as is the FTI which will be the first to diminish. Tests to be performed to rule out hyperthyroidism are summarized in Table 3. The radioactive iodine uptake (RAIU)

TABLE 3
TESTS FOR HYPERTHYROIDISM

Tests	Results
Essential for Diagnosis	
◦ Thyroxine (T_4); resin triiodothyronine uptake (RT_3U); free thyroxine index (FTI)	High
◦ Triiodothyronine (T_3) radioimmunassay (RIA)	High
Helpful in Selected Cases	
◦ Radioactive iodine uptake (RAIU)	High unless iodine-loaded
◦ Thyroid scan	Diffusely enlarged except for toxic nodules or exogenous hormone
◦ Thyroid stimulating immunoglobulins (TSI)*	High but difficult to obtain, some are negative
◦ Thyrotrophin-releasing hormone (TRH) or T_3 suppression	When euthyroid Graves' disease is suspected
Prior to Therapy	
◦ Complete blood count	If thioureas are used
• Pregnancy test for women	If radioiodine ablation used
• Exophthalmometry	Baseline in case of proptosis

*Includes long-acting thyroid stimulator (LATS) and LATS protector.

is performed in hyperthyroid patients primarily as a guide to therapy. Tests to confirm or rule out hypothyroidism are summarized in Table 4.

TABLE 4
TESTS FOR HYPOTHYROIDISM

Tests	Results
Essential for Diagnosis	
• Thyroxine (T_4); resin triiodothyronine uptake (RT_3U); free thyroxine index (FTI)	Low
◦ Thyrotrophin (TSH)	High (primary) and low or normal (secondary)
Helpful in Selected Cases	
• Triiodothyronine radioimmunassay (T_3 RIA)	Low or normal
◦ Thyroid scan	Patchy, partial or absent uptake, rarely normal
◦ Thyrotrophin-releasing hormone (TRH)	For patients with normal TSH
◦ Antithyroid antibodies	High titre antimicrosomal ± antithyroglobulin in Hashimoto's disease
◦ Electrocardiogram	Bradycardia, low voltage, prolonged intervals, flattened or inverted T waves
◦ Serum cholesterol	Elevated, reduced with therapy

OTHER THYROID TESTS

Some thyroid functional abnormalities require stimulation or suppression tests for more accurate definition. These include autonomy of thyroid function without gross hypersecretion and locally autonomous thyroid tissue. Methods also exist of pinpointing the site of the lesion in hypothyroidism, whether it be hypothalamic, pituitary, or thyroid. Although these methods are not routinely available outside the thyroid or nuclear medicine clinic, an understanding of their indications and implications is

useful. A number of ways to further assess thyroid anatomy are briefly listed in Table 5.

TABLE 5
TECHNIQUES FOR EVALUATING THYROID ANATOMY

Techniques	Comments
Scans	
Rectilinear scanner	Best delineation of nodules; less expensive; one-to-one image
Scintillation camera	Less time per scan; allows multiple views; use of ^{99m}Tc and ^{123}I only
Ultrasound	Disappointing; no radiation to patient
Roentgenography	
Conventional Type	Substernal goiters; calcification; metastases may be detected
Biopsy	
Needle	Poor correlation with true histologic diagnosis; no anesthesia risk
Open	No advantage over thyroidectomy
Aspiration	Sometimes useful for cysts

Thyrotrophin-Releasing Hormone

The use of the hypothalamic hormone TRH (Thypinone®) in the diagnosis of thyroid disorders will clarify some of the more obscure cases. The normal role of TRH in thyroid physiology has not been completely established, but it is known to have well-defined effects on the pituitary. An intravenous injection of the synthetic TRH tripeptide brings about a prompt release of pituitary TSH which is blocked by the presence of excess thyroid hormones, since the primary site of feedback control in the thyroid system is in the pituitary rather than in the hypothalamus. The TRH test is thus useful in the diagnosis of hyperthyroidism when blood levels of thyroid hormones are not high enough to clearly confirm thyrotoxicosis. This becomes important in establishing

the entity known as "euthyroid Graves' disease" where ophthalmopathy or simple goiter in the absence of high T_4 or T_3 levels is associated with nonsuppressible RAIU. Here the TSH will not rise after TRH injection because of relatively, but not absolutely, high thyroid hormone concentrations in the circulation. The test is safer and less expensive than the T_3 suppression test, as well as less time-consuming.

In patients with hypothyroidism without high levels of TSH in the serum, the site of the defect in the hypothalamic-pituitary-thyroid axis may be determined by TRH testing. An excessive TSH response means primary hypothyroidism; an absent TSH response indicates a pituitary disorder; and a qualitatively normal but delayed response suggests hypothalamic disease. Unfortunately, some patients with non-primary hypothyroidism may have confusing test results.

The TRH test is quite simple to perform, requiring only 3 blood samples drawn prior to, and at 20 or 30, and 60 minutes following an intravenous dose of 200 to 500 mcg of TRH. A normal response consists of a 3-fold to 5-fold increase in TSH at 20 or 30 minutes. Possible side effects of this test are a transient wave of nausea, or odd taste, which pass rapidly although emesis occasionally occurs. Blood pressure changes, which occur rarely, may be dangerous to certain patients.

Triiodothyronine Suppression Test or Werner's Test

The ability of thyroid hormones to suppress thyroid function by reducing TSH secretion has both diagnostic and therapeutic implications. The phenomenon of thyroid autonomy or independence of TSH can result from stimulation by immunoglobulins as in Graves' disease, or it may be caused by an endogenous lesion, such as an adenoma or an autonomously-functioning nodule in a multinodular goiter. Because not all patients with Graves' disease have thyrotoxicosis, and because autonomous nodules are frequently not hyperactive, the T_3 suppression test helps to confirm these diagnoses. In addition, the test may save the patient from undergoing a long

period of iatrogenic thyrotoxicosis if thyroid hormone treatment is used to shrink the nodule or goiter.

The test may be performed with or without a baseline RAIU and scan although it is preferable for comparison. A dose of 75 to 100 mcg of T_3 (Cytomel®) is given daily for 7 days, followed by a RAIU. A scan is necessary in the case of one or more nodules and is optional in the patient with suspected Graves' disease. The serum T_4 may be measured as an additional indicator of thyroid suppression. The relatively short half-life of T_3 (just over one day) facilitates the withdrawal of the drug, should worsening thyrotoxicosis or angina pectoris ensue. While L-thyroxine may be used in place of T_3, it does have certain drawbacks such as the inability to monitor serum T_4 and the long half-life in case of complications. Its main advantage is the use of a single 3.0 mg dose which eliminates the chance of patient error in taking the medication.

The failure of the 24-hour RAIU to fall below 5 percent after a full week of T_3 suggests Graves' disease in a patient with typical eye findings or a diffuse goiter. Presence of localized uptake of the radionuclide on the scan, even after the RAIU has fallen significantly, indicates an autonomous nodule. If this is the only area which contained isotope prior to suppression with T_3, it is likely that it is an adenoma and that the patient is hyperthyroid.

Thyrotrophin Stimulation

Although rarely done now, the injection of bovine TSH may help to distinguish disorders with diminished TSH release from others which are anatomically similar. Thyrotrophin will cause thyroid tissue suppressed by a toxic nodule to "light up" on scan, thus proving autonomy and hyperfunction in the nodule, and differentiating it from a thyroid with absence of, or thyroiditis in one lobe. Once used to distinguish primary from secondary hypothyroidism, it has been supplanted by the serum TSH assay. It may also be used to assess thyroid function in a patient already on thyroid hormone, or to determine thyroid reserve in euthyroid patients following thyroiditis or therapy for Graves' disease.

An absolute increase in the RAIU of 10 percent or more, or double the baseline value, should occur following 10 units of bovine TSH administered intramuscularly. The major difficulty with this test is the potential for a severe allergic reaction to the foreign protein.

References

1. Magnus-Levy A: Über den respiratorischen Gawechsel unter den Einfluss der Thyroidea sowie unter verschiedenden pathologischen Zustanden. Berlin Klin Wochenschr 32:650-652, 1895.
2. Chaney WC: Tendon reflexes in myxedema; a valuable aid in diagnosis. JAMA 82:2013-2016, 1924.
3. Baumann E: Z Phys Chem 22:1, 1896 cited in editorial Lancet 898, 1971.
4. Kendall EC: J Biol Chem 20:501, 1915 cited in editorial Lancet 898, 1971.
5. Lerman J: Iodine components of blood. Circulating thyroglobulin in normal persons and in persons with thyroid disease. J Clin Invest 19:555-560, 1940.
6. Gross J, Pitt-Rivers R: Identification of 3:5:3'-L-triiodothyronine in human plasma. Lancet 1:439-441, 1952.
7. Barker SB, Humphrey MJ, Soley MH: Clinical determination of protein-bound iodine. J Clin Invest 30:55-62, 1951.
8. Pileggi VJ, Lee ND, Golub OJ et al: Determination of iodine compounds in serum. I. Serum thyroxine in the presence of some iodine contaminants. J Clin Endocrinol Metab, 21:1272-1279, 1961.
9. Murphy BE, Patee CJ: Determination of thyroxine utilizing the property of protein binding. J Clin Endocrinol Metab, 24:187-196, 1964.

COLORECTAL POLYPS

MAJ Robert Ogburn, MC

The colorectal polyp, a commonly seen entity, varies in frequency from 5 to 12 percent in clinical studies and up to 70 percent in autopsy studies with meticulous examination of the colon. /1/ Most commonly, the colonic polyp is clinically asymptomatic, and it is fortuitously encountered in the course of sigmoidoscopic or radiographic examination of the colon for unrelated symptoms.

The clinical significance of the single colorectal polyp, its relationship to the development of colonic cancer, and its proper management have long been subjects of seemingly endless controversy. Fortunately, acceptance of uniform histopathologic terminology and the refinement of fiberoptic colonoscopy as both a diagnostic and therapeutic technique promise to resolve much of the controversy.

The aim of this article is to discuss briefly the histopathology of colorectal polyps and to apply this classification to a description of the clinical presentation and management of colonic polyps. A brief discussion of the multiple polyposis syndromes and the relationship of the solitary polypoid adenoma to colorectal cancer are given.

HISTOPATHOLOGIC CLASSIFICATIONS

The term "polyp" is defined by Stedman's medical dictionary as "a general, descriptive term used with reference to any mass of tissue that bulges or projects outward, or upward, or downward from the normal surface level." Polypoid lesions of the colon exhibit differing

natural histories, depending on their histologic types; it is therefore essential to derive a uniform histopathologic classification of colonic polyps before discussing their significance and management. Morson's classification /2,3/ is gaining increasing acceptance and should help clarify what up to this time has been a confused area (Table 1).

TABLE 1
CLASSIFICATION OF
BENIGN POLYPOID LESIONS OF THE LARGE BOWEL*

Type of Colonic Polyp	Examples
Unclassified	Hyperplastic (metaplastic) polyp
Neoplastic	
Epithelial origin (adenoma)	Tubular adenoma (adenomatous polyp); villotubular adenoma (villoglandular polyp, papillary adenoma, mixed polyp); villous adenoma (villous papilloma)
Nonepithelial origin	Carcinoid; lipoma; leiomyoma; fibroepithelioma
Inflammatory	Benign lymphoid polyp (benign lymphoma); pseudopolyp
Hamartomatous	Juvenile (retention) polyp; Peutz-Jeghers polyp

*Table adapted from Morson and Dawson.[2]

TYPES OF COLONIC POLYPS

Hyperplastic Polyps

The hyperplastic or metaplastic polyp accounts for about 90 percent of all colonic polyps. /4,5/ Nearly always less than 5 mm in size, it appears most frequently in the rectosigmoid area, is often multiple, and has a "dewdrop" appearance with normal mucosal coloration. /2/ The hyperplastic polyp results from a minor imbalance in cell renewal with excessive cell production. There is normal differentiation into goblet and absorptive cells,

and formation of elongated tubules which often show cystic dilatation. Cellular hyperplasia and increased mitosis are confined to the lower portions of the crypts. /4/ It is generally accepted that hyperplastic polyps are asymptomatic and not related to neoplastic polyps or carcinoma. Their only significance lies in the confusion they cause if mistaken for neoplastic polyps. /2/

Neoplastic Polyps

Neoplastic polyps of epithelial origin comprise 9 to 10 percent of all colonic polyps. /5/ They are classified as tubular adenomas (adenomatous polyps) which are usually pedunculated or have a discernible stalk, and villous adenomas, which are lobulated and sessile or broad-based in growth pattern. There appears to be a spectrum of histologic types, with frequently occurring intermediate forms, classified by Morson as villoglandular adenomas. /2,3,7/ In most series, the tubular growth pattern appears more frequently than the villous pattern. The villous adenoma, however, is found more often to contain invasive cancer (Table 2).

TABLE 2
HISTOLOGIC TYPE OF POLYP AND INCIDENCE OF CARCINOMA*

Histologic Type	Total Number	Percent	Number with Malignancy†	Percent
Tubular adenoma	1880	75.0	90	4.8
Intermediate	383	15.3	86	22.5
Villous adenoma	243	9.7	99	40.7

*Table adapted from Morson.[6]
†Defined only in the presence of invasion across the muscularis mucosa.

Under the microscope, all adenomas show the characteristics of true neoplasms, with increased mitosis seen at all levels of tissue. Cell differentiation is incomplete and enlarged hyperchromatic nuclei are present. There is

a variable amount of atypia, and all stages from *in situ* to frankly invasive carcinoma may be present in an adenoma.

Inflammatory Polyps

Benign lymphoid polyps are most commonly found in the lower rectum. They are composed of normal lymphoid tissue and may be up to 3 cm in size. They are usually asymptomatic, sessile, fleshy tumors which have been termed "anal tonsils" and have no neoplastic potential. /2/ Pseudopolyps are seen in inflammatory disorders of the colon (ulcerative colitis, Crohn's colitis, bacterial or amoebic dystentery, schistosomiasis) and consist of islands of normal mucosa which have been undermined by the inflammatory process.

Hamartomas

Juvenile polyps occur primarily during childhood, but may also be seen in adults. They may be multiple, and tend to occur in the rectum or left colon. They are usually under 2 cm in size and present with rectal bleeding or prolapse. Most juvenile polyps are pedunculated and there is a predisposition to autoamputation. /1,5/ Upon microscopic examination they are characterized by cystic dilated epithelial tubules in an abundant stroma of lamina propria with secondary inflammation. These lesions are not neoplastic, and have no malignant potential. /2/

SYMPTOMS

Hyperplastic polyps are essentially always clinically silent, as are most adenomatous polyps less than 1 cm in size. In Welch's series /5/ 46 percent of adenomas larger than 1 cm presented with a mild hematochezia, characterized by small amounts of bright red blood on the surface of the stool and sometimes accompanied by mucus.

Villous adenomas in the left colon or rectosigmoid can be associated with passage of copious amounts of rectal mucus with resulting diarrhea and symptomatic hypokalemia. /5/

Other presenting symptoms are seen with larger polyps. A change in bowel habits due to partial obstruction is occasionally seen with a sigmoid or rectal polyp but occurs more frequently with carcinoma. Some patients develop a low grade intussusception from a large polyp, and present with crampy pain. Low rectal polyps may present with prolapse.

MULTIPLE POLYPOSIS SYNDROMES

Familial Colonic Polyposis

This is a disorder in which large numbers of typical adenomas are found in the colon. Onset of the polyps is usually in the second or third decades of life, with development of colonic carcinoma 5 to 12 years later. /6/ Familial colonic polyposis is an autosomal dominant disorder, with a frequency estimated at 1:8,500 births. Preferred treatment of an affected individual is total proctocolectomy before the inevitable development of colonic carcinoma. Periodic evaluation of other family members at risk is imperative. /1,2,6,8/

Gardner's Syndrome

Gardner's syndrome in which adenomas of the colon are associated with osteomas, dental malformations, and soft tissue tumors (epidermal inclusion cysts, lipomas, fibromas) is an autosomal dominant disorder. Onset of the colonic polyps is usually postpubertal, and follows the other manifestations. Natural history of the colonic adenomas is identical to those formed in familial colonic polyposis, and treatment is the same. An additional risk to the patient with Gardner's syndrome is the development of retroperitoneal fibrosis and sarcomas from the mesenchymal tumors. /1,2,9/

Peutz-Jeghers Syndrome

This syndrome is an autosomal dominant disorder defined by the occurrence of hamartomatous polyps at any level of the gastrointestinal tract associated with melanin spots in a characteristic distribution. The melanin spots are typically found perorally, on oral mucous membranes, and on the hands. Patients with this disorder usually present with gastrointestinal bleeding or small bowel obstruction from intussusception. Risk of carcinoma of the bowel is low and surgery is reserved for refractory bleeding or obstruction. /1,2/ Peutz-Jeghers polyps occur in the small bowel more commonly than in the colon. They appear lobulated and on microscopic examination contain all the normal intestinal elements with a prominent mucosa.

SOLITARY POLYPOID ADENOMA

Incidence

The most common clinically significant problem is that of the solitary polypoid adenoma. Its frequency varies widely, ranging from 1 percent in children at proctoscopy to 70 percent in autopsy studies in which the whole colon is meticulously examined with a hand lens. A consideration of only those polyps 1 cm or larger yields an incidence of polyposis of 5 to 12 percent. /1,5/ It is well recognized that the incidence of polyps increases with age, reaching a peak in the seventh decade. /1/ It has classically been taught that most polyps are located in the rectosigmoid, within reach of the sigmoidoscope, but necropsy studies which consider only adenomas show that they are fairly evenly distributed throughout the colon. In about one-third of the patients with an adenoma there is at least one additional polyp present in the colon. /1,2,5/

The increasing incidence of adenomas with advanced age and the increased incidence of both adenomas and carcinomas in groups consuming a diet high in animal fat and low in

fiber content suggest that prolonged exposure to environmental carcinogens may be important in the pathogenesis of adenomas. /1/ Additional evidence suggesting a role of environmental carcinogens comes from animal studies. In the rat, administration of dimethylhydrazine and a number of other carcinogens can produce a full range of colonic neoplasms, from adenomas to invasive carcinomas. /14/

DIAGNOSIS

The standard barium enema is quite insensitive in demonstrating polyps. In Welch's study /5/ polyps larger than 1 cm were seen in 3 percent of standard barium enema examinations; this figure rose to 10 percent with an air contrast study. Williams /10/ compared conventional barium enema and air contrast (Malmo) barium enemas with colonoscopy for demonstration of polyps (Table 3). In this series, a demonstrated lesion on barium enema could not be seen by colonoscopy on only two occasions. In another series comparing colonoscopy with air contrast barium enema, 166 polyps were diagnosed radiographically and an additional 118 were seen at colonoscopy. Twenty-one of these exceeded 1 cm in size; radiographic abnormalities were successfully evaluated at colonoscopy in 99.6 percent of cases. /11/

TABLE 3

DEMONSTRATION OF COLONIC POLYPS*

	Conventional Barium Enema			Air Contrast (Malmo) Barium Enema		
Polyp Size	X-ray	Colonoscopy	Seen At X-ray *(%)*	X-ray	Colonoscopy	Seen At X-ray *(%)*
≤ 5	6	55	18	110	147	78
6-10	6	10		42	48	
11-20	15	20	77	74	75	98
> 20	18	23		21	22	
Totals	45	108	42	247	292	85

*Table adapted from Williams, Hunt, Loose, et al.[10]

From the above studies, it can be concluded that conventional barium enema fails to demonstrate a significant proportion of polyps larger than 1 cm, air contrast barium enema demonstrates 80 to 98 percent of these lesions, and colonoscopy in skillful hands not only demonstrates additional potentially significant polyps, but can completely remove most of them.

POLYP–CANCER CONTROVERSARY

The controversy over the relationship of the adenomatous polyp to carcinoma of the colon is a continuing one. Some authorities /1,15/ believe that adenomatous polyps have a low malignant potential and that most carcinomas arise *de novo* in normal mucosa. They feel that the frequently noted association of adenomatous polyps and carcinoma in the same colon may reflect the fact that both are caused by the same hereditary and environmental factors, and that a lesion remains either benign or malignant from its inception. /1,15/ Other authorities /4/ feel that most, if not all, colonic carcinomas arise by malignant transformation in an adenoma; these authorities vociferously deny "the myth of *de novo* carcinoma". The general consensus of several authorities /3-7,13,15,18,19/ with few exceptions is that the common adenomatous polyp is probably a premalignant lesion, although rigid scientific proof of this is lacking.

Direct clinical evidence of the sequential development of carcinoma in a previously-benign adenoma is impossible to obtain; complete evaluation of a polyp for focal cancer requires total excisional biopsy, a process which precludes observation *in situ*. However, a considerable body of indirect evidence suggests that adenomatous polyps are in fact the precursors of colon carcinoma. These include:

- Typical adenomatous polyps which are seen in familial colonic polyposis. These are premalignant lesions which become sources of multifocal carcinoma if observed for 5 to 10 years. /5,12/

- The peak incidence of polyposis in the general population antedates the peak incidence of carcinoma by 5 to 10 years. /3/

- On rare occasions, apparently benign polyps have repeatedly been observed *in situ* with a high incidence of occurrence of carcinoma developing at the same site in 5 to 10 years. /3/

- Examples of small (less than 5 mm) intramucosal carcinomas have not been reported in normal non-adenomatous mucosa, but are commonly seen in adenomas. /4/

- Experimental carcinogens produce both typical adenomas and carcinomas in animals. /13/

- One-third of all colons removed for cancer have one or more additional benign adenomas; the distribution of cancers and adenomas is similar. /5,16/

- A prospective study of 12,000 patients over a 16-year period in which all polypoid lesions seen on repeated proctoscopic examinations were removed, resulted in an 85 percent decrease in the predicted incidence of colonic carcinoma. /17/

The risk of carcinoma occurring in an adenomatous polyp has been found to be most reliably predicted by the size of the polyp. Morson's data are representative (Table 4) in that the risk of cancer in an adenoma less than 1 cm in size is about 1 percent, rising to about 50 percent for polyps larger than 2 cm.

TABLE 4
SIZE OF ADENOMAS AND RISK OF CARCINOMA*

Size of Tumor *(cm)*	Total Number	Number With Malignancy	Percent
< 1	1,479	19	1.3
1-2	580	55	9.5
> 2	430	198	46.0

*Table adapted from Morson.[6]

MANAGEMENT

With the advent of the colonoscopic polypectomy, the management of polypoid adenomas has become much less controversial. This technique makes polypectomy easier, safer, and less costly to perform than the surgical approach. Evaluation should start with proctoscopy; radiographic evaluation for suspected colonic polyps should include an air contrast barium enema. All demonstrated polyps accessible to the proctoscope or colonoscope should be removed. Radiographically-demonstrated polyps not accessible to the colonoscope should be surgically removed if (a) they are 2 cm or larger in size; (b) 1 to 2 cm in size and the patient is a good surgical candidate; and (c) radiographic appearance, biopsy, or cytology suggest carcinoma. Small, inaccessible polyps should be followed by barium enema at 6 to 12 month intervals, and, if they enlarge, should be surgically removed.

FOLLOWUP

A study of patients after removal of adenomatous polyps /20/ has shown recurrent polyps on follow-up in 30 percent of all patients; polyps recurred at a frequency of 16 times greater than predicted during the first year, but approximated that of the general population after two years. /20/ Based on this study, postpolypectomy follow-up should ideally include proctoscopy and either colonoscopy or air contrast barium enema at yearly intervals for at least several years.

There has been some uncertainty about proper management of the patient with a microscopic focus of cancer in a removed polyp. Based on the series by Wolff and Shinya /21/ complete polypectomy is adequate treatment for *in situ* cancer in a polyp. If there is invasive cancer in the head of a polyp, complete polypectomy is probably adequate treatment unless tumor is close to the plane of resection, or the tumor is highly undifferentiated or present in the lymphatics. If there is a question about the adequacy of treatment, Wolff and Shinya recommend laparotomy with segmental resection of the affected colon. /21/

CONCLUSION

The approach to colonic polyposis has been simplified by improved diagnostic and therapeutic techniques. More uniform histologic classification should help clarify the natural history of the polypoid adenoma and its relationship to the development of colon cancer. Present evidence suggests that the colonic adenoma is a premalignant

lesion, and that most colon cancer probably arises in pre-existing adenomas.

Colon cancer is the most common visceral neoplasm in the United States today, with 99,000 new cases and 48,000 deaths per year. /18/ The possibility of preventing a large proportion of these tumors by vigorous removal of all colonic adenomas presents an exciting therapeutic challenge.

References

1. Haubrich WS, Berk JE: Benign tumors of the colon and rectum, in Bockus HL (ed): Gastroenterology. Philadelphia, WB Saunders, 1976
2. Morson BC, Dawson IMP: Gastrointestinal Pathology. London, Blackwell Scientific Publications, 1972
3. Morson BC: Genesis of colorectal cancer. Clin Gastroenterol 5:505-525, 1976
4. Fenoglio CM, Lane N: The anatomic precursors of colorectal carcinoma. JAMA 231:640-642, 1975
5. Welch CE, Hedberg SE: Polypoid lesions of the gastrointestinal tract. Major Problems in Clinical Surgery, ed 2. Philadelphia, WB Saunders, 1975, vol 2
6. Morson BC: Relationship of polyps to cancer of the colon and rectum. Read before the Digestive Disease Week postgraduate course on gastrointestinal cancer, San Antonio, 1975
7. Jackson BP: Adenomas of the colon and rectum: Histopathology and management. Dis Colon Rectum 17:656-657, 1974
8. McConnell RB: Genetic aspects of gastrointestinal cancer. Clin Gastroenterol 5:483-502, 1976
9. Watne AL, Johnson JG, Chang CH: The challenge of Gardner's syndrome. Cancer 19:266-275, 1969
10. Williams CB, Hunt RH, Loose H, et al: Colonoscopy in the management of colon polyps. Br J Surg 61: 673-682, 1974
11. Wolff WI, Shinga H, Geffen A, et al: Comparison of colonoscopy and the contrast enema in five hundred

patients with colorectal disease. Am J Surg 129:181-186, 1975
12. Levin B, Riddell RH, Kirsner JB: Management of precancerous lesions of the gastrointestinal tract. Clin Gastroenterol 5:827-853, 1976
13. Sherlock P, Lipkin M., Winawer SJ: Predisposing factors in carcinoma of the colon. Adv Intern Med 20:121-150, 1975
14. Bralow SP, Weisburger JH: Experimental carcinogenesis in the digestive organs. Clin Gastroenterol 5:522-542, 1976
15. Castleman B, Krickstein H: Carcinoma arising in adenomatous polyps of the colon is greatly exaggerated, in Ingelfinger FJ (ed): Controversy in Internal Medicine, Philadelphia, WB Saunders, 1966
16. Ekelund G, Lindstrom C: Histopathological analysis of benign polyps in patients with carcinoma of the colon and rectum. Gut 15:654-663, 1974
17. Gilbertson VA, Knatterud GL, Lober PH, et al: Invasive carcinoma of the large intestine: A preventable disease? Surgery 57:363-365, 1975
18. Corman ML, Veidenheimer MC, Coller JA: Recent thoughts on the development of colorectal cancer. Med Clin North Am 59:347-361, 1975
19. Whitehead R: Rectal polyps and their relationship to cancer. Clin Gastroenterol 4:545-561, 1975
20. Henry LG, Condon RE, Schulte WJ, et al: Risk of recurrence of colon polyps. Ann Surg 182:511-515, 1975
21. Wolff WI, Shinya H: Definitive treatment of 'malignant' polyps of the colon. Ann Surg 182: 516-525, 1975

INDIGESTION: A disease which the patient and his friends frequently mistake for deep religious conviction and concern for the salvation of mankind. As the simple Red Man of the western wild put it, with, it must be confessed, a certain force: *"Plenty well, no pray; big bellyache, heap God".*

Ambrose Bierce (1842 to 1914[?])
The Devil's Dictionary.

THE RATIONAL USE OF THERAPEUTIC HEAT

LTC Donald H. See, MC

Man has a long history of applying heat to relieve the discomfort that accompanies numerous disorders such as painful arthritic, musculoskeletal, and traumatic conditions. A common attitude that "heat is good" for almost any painful condition has led to widespread, indiscriminate use and abuse of heat by layman and health professional alike. Heat is frequently used without specific therapeutic goals and with little understanding of potential physiologic responses. The intensity, duration, frequency, and type of heating are usually applied empirically. Several physiologic responses to heat application have been adequately studied and proven to be therapeutically beneficial. /1/ Some of these responses cannot be produced by means such as drugs or exercise; heat has the additional advantages of localized application and controlled dosimetry.

The approximate range of tissue temperature considered to be therapeutically beneficial is relatively narrow, extending from 40° to 45° C. /1/ Tissue destruction begins with temperatures above this range. The margin of safety between effective temperature and potentially dangerous temperature is narrow; techniques and precautions in application are, therefore, of great importance. For purposes of discussion in this paper, the application of heat will refer to heating in the therapeutic range of 40° to 45° C. Heating in the lower portion of the range will be considered mild heating and heating in the upper portion will be considered vigorous heating.

Physiologic Responses

A rational basis for heat application can be developed if one understands the nature of the pathologic condition

and is aware of the physiologic responses to heat (Table 1). Specific goals should be developed consistent with the pathology and the potential for heat to alter it in a significant therapeutic manner.

TABLE 1
PHYSIOLOGIC RESPONSES TO HEAT

- Increase in pain threshold
- Increase in blood flow
- Increase in extensibility of collagen fibers
- Decrease in joint stiffness
- Decrease in muscle spasm

Heat acts directly on peripheral nerve fibers and free nerve endings in the tissues, producing an increase in the pain threshold /2,3/; this can be accomplished by heating the area directly or heating the nerve supply to the area. Utilization of this analgesic effect undoubtedly accounts for the vast majority of heat applications. Heat applied for the relief of pain is also the basis for its frequent misuse. Heat all too often is used for excessively long periods of time or at excessive temperatures, producing edema and tissue destruction. A common example is the practice of falling asleep with an electric heating pad and awakening with second or third degree burns. Heat increases the tissue metabolic rate, producing an increased quantity of metabolites; this, in turn, causes dilatation of arterioles and capillaries and increases local blood flow /4,5,6/, capillary permeability, and diffusion, leading to edema. Preexisting edema is therefore likely to be aggravated. Many of the responses of acute inflammation are produced by heating which increases the extensibility of collagen fibers. /7,8/ Collagen at normal body temperature primarily exhibits elastic properties and minimal viscous flow; that is, it returns to its original length after stretch is applied. At therapeutic temperatures the viscous flow becomes predominant, tension relaxes, and stretching of fibrous tissues during this condition results in residual elongation. This remarkable effect is utilized clinically by combining heating with an appropriate exercise program of stretching. Organized connective tissue found in tendons, ligaments, and joint capsules is comprised primarily of compact collagen fibers. Dense connective tissue as seen in

muscle and organ capsules and fascial planes contains meshworks or loose bundles of collagen fibers. Trauma, immobilization, edema, and decreased circulation to connective tissue result in thickening, shortening, adhesions, scars, and fibrosis which, in large part, is formation and maturation of collagen fibers. The potential for regaining and maintaining mobility previously limited by joint and soft tissue contractures is significant if the involved connective tissue can be heated to therapeutic levels and stretched while still heated. The benefit of increased extensibility can be obtained only when it is combined with a carefully planned program of stretching. Heat application without exercise cannot improve mobility. Heating the joints of patients with rheumatic disease produces both *objective* and *subjective* relief of joint stiffness. /9/ Passive resistance to finger flexion and extension as measured and displayed with a hysteresis loop reveals a significant *objective* decrease in joint stiffness by heat application. The patient's *subjective* complaints of stiffness correlate with changes in the viscoelastic properties of the joints. Decrease in muscle spasm may be obtained by heat therapy. /10/ Heat has a direct effect on gamma fiber activity with resultant decrease in sensitivity of the muscle spindle to stretch. Protective muscle spasm is sometimes seen to accompany acute painful conditions, such as acute low back pain. The presence of muscle spasm is, however, commonly overdiagnosed.

Contraindications

There are probably no absolute, unequivocal contraindications to heat application. Under several conditions, its use is generally contraindicated and the potential benefits rarely outweigh the potential hazards (Table 2).

TABLE 2
CONTRAINDICATIONS TO HEAT APPLICATION

- In areas where circulation is impaired
- In areas where sensation is impaired
- In hemorrhagic disorders
- In impaired state of awareness
- In areas over malignancies

AD-A044 630 LETTERMAN ARMY MEDICAL CENTER SAN FRANCISCO CALIF F/G 6/5
AMBULATORY INTERNAL MEDICINE CARE SYMPOSIUM, PRESENT CONCEPTS I--ETC(U)
1977 T J EGAN, N Z SANDERS, C E SWEE

UNCLASSIFIED NL

2 OF 2
AD A044630

END
DATE FILMED
10-77
DDC

Local heating of ischemic tissue increases the metabolic demand beyond the ability of the vasculature to supply sufficient blood, with resultant tissue necrosis. The presence of peripheral vascular disease is the most common contraindication to the application of heat. Heating areas that are anesthetic or have significantly impaired sensation is hazardous. Dosimetry of heat application is generally not highly accurate and much reliance in preventing excessive temperatures is placed on the patient's ability to perceive the sensations of warmth and burning pain. A patient with combined peripheral neuropathy and peripheral vascular disease such as often occur in diabetes mellitus would be at high risk for heat application. Vigorous heating is generally contraindicated in the presence of a hemorrhagic diathesis which may produce or accelerate bleeding. Heat application is contraindicated during the first 48 to 72 hours following trauma if bleeding or edema are present. The patient with significantly impaired awareness secondary to any cause cannot be safely treated with heat. Heat should not be applied when there is a malignancy in the treatment field; the increased blood flow may increase the potential for metastasis and the rate of tumor growth can be accelerated by moderate elevation of temperature.

Factors Determining the Extent of Physiologic Response

The two major determinants of the extent of biologic response to heat application are (1) the *level* of tissue temperature elevation and (2) the *duration* of the tissue temperature elevation. Core temperature is 37° C; the therapeutic range in which the previously-described physiologic responses occur is approximately 40° to 45°. Typical skin surface temperatures are: trunk 31° C, hand 30° C, foot 27° C. Elevation of superficial structures to core temperature falls significantly below the therapeutic range and requires further increases for beginning biologic responses. It is probably that because of lack of knowledge, poor technique, and inappropriate modality selection, the majority of heat applications never produce biologically effective temperature levels at the site of the pathology. Only a placebo effect would be anticipated at these

subtherapeutic levels. The duration of heating must be considered in reference to the total time the heat is applied and the portion of that time during which therapeutic levels are maintained. Most modalities require approximately 10 minutes to generate temperatures in the therapeutic range. The minimal effective exposure time after appropriate temperatures have been reached is approximately 5 minutes. Complete results are achieved after exposures of approximately 30 minutes. Based on these factors, the total treatment duration is usually 20 to 30 minutes. Heating beyond 30 minutes is considered excessive and may produce undesirable effects, particularly edema.

Appropriate therapeutic goals are developed by considering the underlying pathology and the spectrum of desirable physiologic responses that can be achieved. The next step is to select a heating modality that is capable of producing the desired responses at the site of the pathology.

MODALITIES PRODUCING SUPERFICIAL HEAT

A number of sources that can produce vigorous heating in the skin and subcutaneous tissues are available; at best, they result in only minimal heating of intermediate tissues and have no effect on deep structures.

Hydrocollator Packs

These commonly-used packs contain a silica gel which absorbs a large volume of water. The pack is heated in a water bath maintained at 79° C and applied with terry cloth between the pack and the skin. Skin temperature is elevated rapidly, subcutaneous tissues are heated only moderately, and deeper tissues, including muscles, are not heated.

Infrared Lamp

Electromagnetic radiation in the visible and infrared spectrum is absorbed by tissue and converted to heat energy. The superficial tissues can be heated vigorously but there is little effect on deeper structures. A 250 watt Mazda® heat

lamp with a reflector is a convenient and inexpensive modality ideal for home use. Joints with minimal covering such as those in the hand can be vigorously heated with luminous heat, making this ideal for many arthritic conditions. /11/

Paraffin Bath

Paraffin is melted and mixed with mineral oil and maintained at a temperature of approximately 52° C. The hand or foot is repeatedly dipped into the paraffin or kept submerged.

Hot Water

Heated water in tubs and tanks carries heat to the skin by convection. This technique is most beneficial on burns, ulcers, and wounds when combined with its debriding action using a water agitator. The extremity treated must necessarily be in a dependent position, e.g., an arm or a leg dangling in a whirlpool tank; unfortunately, any tendency for dependent edema will thereby be enhanced.

Electric Heating Pad

A common source of misuse of heat, the heating pad typically is taken to bed and applied for excessive lengths of time. Thermostatic control is often poor, the patient falls asleep, and burns occur over bony prominences where there is relative ischemia.

MODALITIES PRODUCING DEEP HEAT

Three diathermy or "deep heating" modalities are available: (1) shortwave diathermy, which converts high frequency electrical current into heat; (2) microwave diathermy, which converts electromagnetic radiation into heat; and (3) ultrasound application, in which sound waves are converted into heat energy.

Shortwave Diathermy

Shortwave produces selective heating of subcutaneous tissues and superficial muscle layers. /1/ More deeply lying muscles and joints are not heated. Metallic implants such as prosthetic joints, plates, and pacemakers may be selectively heated and high temperatures may develop. Shortwave diathermy is, therefore, contraindicated in the presence of metallic implants. Including the eye in the treatment field is contraindicated because of potential formation of cataracts.

Microwave Diathermy

Microwave is electromagnetic radiation similar to radar. The waves are also similar to light in that they can be reflected, scattered, refracted, and absorbed. The commercially-available generators emit a frequency of 2,456 megahertz (MHz) which produces selected heating in subcutaneous tissues and superficial muscle similar to the temperature distribution of shortwave diathermy. /1/ Application to the eye and in the presence of metallic implant is contraindicated.

Ultrasound Application

Ultrasound is an acoustic vibration delivered at frequencies of 0.8 and 1.0 MHz, far above the range perceived by the human ear. Therapeutic ultrasound consists of beaming high frequency sound waves into tissues where they are absorbed and converted to heat energy. /1/ Frequencies under 17,000 hertz are considered sound and those above this level are designated ultrasound. The relative protein content of a tissue determines the acoustic impedance and, in turn, the amount of ultrasound absorption. Ultrasound is selectively absorbed at the interface between tissues of different acoustic impedance, such as the muscle-bone interface. This pattern of absorption fortunately produces selective heating in certain structures that are in need of treatment such as a joint capsule, synovium, and ligaments. /12,13,14/ Other selectively heated structures

are scars within soft tissues, myofascial interfaces, tendons, and tendon sheaths. Proper ultrasound application to these deep structures, combined with appropriate exercise, allows effective mobilization of joint and soft tissue contractures. Ultrasound can be safely used in the presence of metallic implants. It is, however, contraindicated in the presence of total joint replacement implants because the coefficient of absorption and the acoustic impedance of the polyethylene glue have not as yet been investigated. Ultrasound is unique in that it passes through skin, subcutaneous tissue, and muscle with minimal absorption and is subsequently absorbed and converted to heat in the deeper structures.

All superficial heating sources produce essentially the same temperature distribution to include small joints such as those of the hand that are covered only by skin and a thin layer of subcutaneous tissue. Shortwave and microwave are similar, producing therapeutic levels only to the depth of relatively superficial muscle and not in deep lying muscle or joints.

Table 3 compares the heating pattern that can be generated in various tissues by the commonly available heating modalities.

DEEP HEATING OF ACUTELY INFLAMED JOINTS

It has long been recognized that applying deep heat to acutely inflamed joints frequently exacerbates the symptoms. Specialists in physical medicine have generally recommended application of only mild, superficial heat, if any, in this situation. Feibel /15/ calls attention to a need to reappraise the use of the deep heating modalities which produce significant temperature elevation in inflamed joints. By contrast, superficial heating for relatively short periods has been shown to *decrease* joint temperatures, apparently by shunting blood from deep to superficial structures. /16/ Recent *in vitro* studies by Harris, et al /17/ have shown that the activity

TABLE 3
RELATIVE HEATING PATTERN OF VARIOUS MODALITIES

Modality	Tissue or Structure to Be Heated					
	Skin	Subcutaneous Tissue	Small Joints*	Superficial Muscle	Deep Joints	Deep Muscle
Modalities producing superficial heat						
Hydrocollator pack	++	++	++	o	o	o
Infrared lamp	++	++	++	o	o	o
Paraffin wax	++	++	++	o	o	o
Hot water	++	++	++	o	o	o
Heating pad	++	++	++	o	o	o
Modalities producing deep heat						
Shortwave diathermy	++	++	++	++	o	±
Microwave diathermy	++	++	++	++	o	±
Ultrasound	o	o	++	±	++	±

*Relatively superficial joints.
Symbols used in the Table: ++ = Heating in therapeutic range possible; ± = Variable unpredictable effect, therapeutic range not often achieved; o = No significant heating effect.

of collagenase, which can destroy articular cartilage, is four times greater at temperatures in rheumatoid knees (36° C) than at temperatures in normal knees (31° C). Hollander /18/ has indicated that these data provide a better rationale for the use of cold or superficial heat over inflamed joints.

CONCLUSION

Application of therapeutic heat can produce several significant physiologic responses that may be clinically useful if properly matched to the pathological condition. The therapeutic heating range is relatively narrow; temperatures below the range are ineffective and temperatures above it can produce tissue destruction. Heat, if indicated, should generally be applied vigorously and for relatively short periods of time. Further elevations of temperature of acutely inflamed joints should be considered potentially harmful.

References

1. Lehmann JF: Diathermy, in Krusen FH, Kottke FJ, Ellwood PM (eds): Handbook of Physical Medicine and Rehabilitation, ed 2. Philadelphia, WB Saunders Co, 1971.
2. Lambert EH, Treanor WJ, Herrick JF, et al: Effect of heat and ultrasound on conduction in bullfrog nerve. Fed Proc 10:78, 1951.
3. Lehmann JF, Stow RW: Pain threshold measurements after therapeutic application of ultrasound, microwaves, and infrared. Arch Phys Med 39:560-565, 1958.
4. Abramson DI, Burnett C, Bell Y, et al: Changes in blood flow, oxygen uptake and tissue temperature produced by therapeutic physical agents. I. Effects of ultrasound. Am J Phys Med 39:51-62, 1960.

5. Bickford RH, Duff RS: Influence of ultrasonic irradiation on temperature and blood flow in human skeletal muscle. Circ Res 1:534-538, 1953.
6. Kemp CR, Paul WD, Hines HM: Studies concerning the effect of deep tissue heat on blood flow. Arch Phys Med 29:12-17, 1948.
7. Lehmann JF, Masock AJ, Warren CG, et al: Effects of therapeutic temperatures on tendon extensibility. Arch Phys Med 51:481-487, 1970.
8. Gersten JW: Effect of ultrasound on tendon extensibility. Am J Phys Med 34:362-369, 1955.
9. Backlund L, Tiselius P: Objective measurement of joint stiffness in rheumatoid arthritis. Act Rheum Scand 13:275-288, 1967.
10. Fischer E, Solomon S: Physiological responses to heat and cold, in Licht S (ed): Therapeutic Heat. New Haven, Elizabeth Licht, 1958.
11. Lehman JF, DeLateur BJ: Heat and cold in the treatment of arthritis, in Licht S (ed): Arthritis and Physical Medicine. New Haven, Elizabeth Licht, 1969.
12. Lehmann JF, DeLateur BJ, Stonebridge JB, et al: Therapeutic temperature distribution produced by ultrasound as modified by dosage and volume of tissue exposed. Arch Phys Med 18:662-666, 1967.
13. Lehmann JF, DeLateur BJ, Warren CG, et al: Heating of joint structures by ultrasound. Arch Phys Med 49:28-30, 1968.
14. Lehmann JF, Warren CG, Scham SM: Therapeutic heat and cold. Clin Orthop 99:207-245, 1974.
15. Feibel A, Fast A: Deep heating of joints: A reconsideration. Arch Phys Med 57:513-514, 1976.
16. Horvath SM, Hollander JL: Intra-articular temperatures as measure of joint reaction. J Clin Invest 28:469-473, 1949.
17. Harris E Jr, McCroskery PA: Influence of temperature and fibril stability on degradation of cartilage collagen by rheumatoid synovial collangenase. N Engl J Med 290:1-6, 1974.
18. Hollander JL: Collagenase, cartilage, and cortisol. N Engl J Med 290:50-51, 1974.

"The pain which is intolerable carries us off;
but that which lasts a long time is tolerable".

Democritus (5th to 4th Cent. B.C.)
Quoted by Marcus Aurelius in
Meditations, VII 33.
(Tr. by G. Long)

INFECTIOUS MONONUCLEOSIS

MAJ Paul J. Killian, MC

Infectious mononucleosis (IM) is an acute and usually self-limited disease characterized by fever, pharyngitis, adenopathy, atypical lymphocytosis, and the development of transient heterophil and persistent Epstein-Barr virus (EBV) antibody. It occurs primarily in the 17- to 25-year-old age group and is well recognized at our college campuses. Indeed, the disease often carries a favorable social stigmata because of its proposed association with intimate oral contact. /1/ Infectious mononucleosis ranks second only to acute respiratory illnesses as a cause of college infirmary admissions. /2/ The importance of this illness among military personnel has only recently been appreciated. Hospital admission rates for IM among the uniformed services range from 140 to 228 per 100,000 persons. /3/

ETIOLOGY AND EPIDEMIOLOGY

Infectious mononucleosis is caused by the EBV, a member of the herpes group. The data supporting this statement come from epidemiologic and immunologic studies. Hallee et al in a prospective study of the 1969 entering class at West Point showed that no cases of IM developed in the 890 cadets with EBV antibody on admission. There were 437 cadets negative for EBV antibody on admission; 201 or 46 percent became positive during the next 4 years. Of these 201 seroconverters, only 26.4 percent manifested heterophil positive clinical IM. This is much lower than was found at English schools /4/ and Yale University /5/ where percentages of seroconverters developing clinically obvious IM were 45 percent and 74 percent, respectively. Differences have been attributed to various patterns of motivation in students to seek medical care. /6/ Evans et al /7/ in seroepidemiologic

studies of IM have found that EBV antibody is consistently present in heterophil positive IM. Antibody to EBV is absent prior to illness and appears during the illness in a rising titer. The EBV antibody is of the IgM class in the acute phase. This persists for several months; then IgG appears and is detectable for life. Diehl et al /8/ have demonstrated EBV by immunofluorescence and electron microscopy from cultures of peripheral leukocytes of patients with IM. Miller et al /9/ have recovered EBV in pharyngeal secretions of a high percentage of patients with IM. Finally, an illness resembling IM has occurred with transmission of EBV by blood transfusion. /10/

CLINICAL MANIFESTATIONS

Infectious mononucleosis has an incubation period of 4 to 7 weeks. /11/ It is ushered in by a prodrome of general malaise, headache, and fatigability. The headache is mild and rarely lasts longer than 3 or 4 days. The complete clinical illness becomes manifest over the next 7 to 14 days and is characterized by adenopathy, fever, and pharyngitis in over 85 percent of cases. The Table /12/ gives the relative percentages of the various signs and symptoms found in IM.

A sore throat is one of the earliest features to appear after the prodrome. In most patients, it subsides by the second week. The tonsils are covered with a gray, shaggy, membranous exudate unlike that present in follicular tonsillitis and they do not bleed when the exudate is removed. /12/ The differential diagnoses include streptococcal infections, Vincent's angina, and diphtheria. On rare occasions, respiratory embarrassment can occur secondary to tonsillar hypertrophy or pharyngolaryngeal edema. Erythematous palatal spots may develop at the junction of the hard and soft palate. Although suggestive, they are not pathognomonic.

TABLE
SIGNS AND SYMPTOMS OF INFECTIOUS MONONUCLEOSIS

Signs	Percentage	Symptoms	Percentage
Adenopathy	100	Malaise and fatigue	95 to 100
Fever	80 to 95	Sweats	80 to 95
Pharyngitis	65 to 85	Sore throat, dysphagia	80 to 85
Splenomegaly	50 to 60	Anorexia	50 to 80
Bradycardia	35 to 50	Nausea	50 to 70
Periorbital edema	25 to 40	Headache	40 to 70
Palate enanthem	25 to 35	Chills	40 to 60
Liver and spleen tenderness	15 to 30	Cough	30 to 50
Hepatomegaly	15 to 25	Myalgia	12 to 30
Rhinitis	10 to 25	Ocular muscle pain	10 to 20
Jaundice	5 to 10	Chest pain	5 to 20
Rash	3 to 6	Arthralgia	5 to 10
Pneumonitis	3	Photophobia	5 to 10

Adenopathy is characterized by symmetrical, discrete, non-tender nodes, posterior cervical involvement being the most common. Hilar adenopathy occurs in less than 1 percent of patients. Nodal enlargement subsides in the third week. /13/ Splenomegaly is present in 50 to 60 percent of patients and is greatest during the second and third weeks. It spontaneously subsides but can remain for months. Although splenic rupture can be fatal, it rarely occurs.

Hepatomegaly is less frequent but mild tenderness is sometimes present. Five to 10 percent of patients develop jaundice. Fever ranging from 100° to 103°F normally is present for several days but subsides in 2 weeks. Three to 5 percent of patients spontaneously develop a faint erythematous maculopapular rash. Eighty to 90 percent of patients with IM, however, develop a rash upon exposure to ampicillin. /14/

Neurological complications fortunately are rare but, if present, can be quite severe. /15/ Encephalitis, meningitis, meningoencephalitis, mononeuritis, polyneuritis, and Guillain-Barre syndrome have all been reported. The cerebrospinal fluid in 25 percent of patients shows increased protein, pressure, and pleocytosis.

LABORATORY FEATURES

Leukocyte count and examination of the peripheral blood smear are essential steps in the early diagnosis of IM. Leukocytosis is present and consists primarily of a relative and absolute lymphocytosis. The total count tends to run between 10,000 mm^3 and 20,000 mm^3 but counts as high as 50,000 mm^3 have been reported. It reaches its peak in the third week and returns to normal in the fourth and fifth weeks. Ten to 20 percent of the lymphs are atypical, large cells with horseshoe-shaped nuclei and vacuolated, foamy cytoplasm. Atypical lymphocytes are not pathognomonic and can be found in infectious hepatitis, drug reactions, and viral infections.

Thrombocytopenia is rarely of clinical consequence; only 0.2 percent develop severe thrombocytopenia. More commonly, 50 percent of patients have platelet counts of 100,000 mm^3 to 150,000 mm^3. /13/ Evaluation of these patients supports a peripheral destruction as the underlying mechanism of thrombocytopenia. Bone marrow examination reveals increased megakaryocytes.

Hemolytic anemia is seen in 3 percent of patients. /13/ It is mild in nature and occurs 1 to 2 weeks after onset. Seventy percent have a positive Coombs test and some patients have increased antibodies. Elevated liver function tests are seen quite commonly. Jaundice, however, is rare. The alkaline phosphatase-bilirubin dissociation is considered useful in supporting the diagnosis of IM. /16/

SEROLOGIC DIAGNOSIS

Patients with IM develop a heterophil antibody of the IgM class that agglutinates sheep red blood cells. Heterophil antibodies have the capability of reacting with antigens entirely different from those which stimulated their production. They can be found in low titer (1:56) in normals and are present in titers as high as 1:224 in patients with Hodgkin's disease, leukemia, and serum sickness. Because of this nonspecificity, the Paul-Bunnell test /17/ is a presumptive test. It should be confirmed with a differential test /18/ which takes advantage of two features peculiar to the sheep cell agglutinins in IM. First, the antibody is not of the Forssman type and thus it is not completely removed by Forssman's antigen (guinea pig kidney). Second, the antibody is completely absorbed with beef erythrocytes. Therefore, the characteristic findings in a patient with IM are positive agglutinin titers, incomplete guinea pig absorption, and complete absorption with beef erythrocytes. Some authors /19/ consider any titer after guinea pig tissue absorption diagnostic, but a titer of 1:28 is definitely diagnostic. The antibody found in serum sickness, for example, is of the Forssman type and is removed by guinea pig kidney. This heterophil antibody is found in 75 percent of patients with IM during the first week, and in nearly all patients by the third week. Thus, if clinically indicated, an initial negative test should be repeated in 2 to 3 weeks.

The heterophil agglutinins are also detectable by horse erythrocytes. In fact, the horse erythrocytes are more sensitive to agglutination than sheep erythrocytes. The commonly used Monospot test, which has a high degree of accuracy and good correlation with the differential test, takes advantage of this sensitivity and of the incomplete absorption with guinea pig kidney; Britta /20/ showed the Monospot to be positive in all patients in whom heterophil

antibody remained after guinea pig kidney. There were no false negatives. False positives have been reported in 5 to 14 percent of sera with other heterophils. Some of these, however, were apparent *false positives as immuno*-fluorescence for EBV antibody showed that some of these patients did indeed have IM. Because of the sensitivity of horse erythrocytes, the Monospot detects titers which are normally not considered positive. This also explains the prolonged positivity of these agglutinins which have been observed in some patients. In summary, the Monospot test has no false negatives. It does have occasional false positives which can be excluded by specific EBV antibody tests.

TREATMENT

Therapy for the most part is symptomatic. Antibiotics have no role unless an infection is clearly defined. Because of the danger of splenic rupture, strenous physical activity and contact sports should be avoided.

Steroids are useful in life-threatening circumstances. This is especially true in cases of respiratory obstruction, central nervous system involvement, thrombocytopenia, hemolytic anemia, and myocarditis. The role of steroids in other situations is not clear. Several studies, /21-23/ not all controlled, have shown that steroids do decrease the duration of fever and hasten the return to full-time activities. Whether or not treatment of this viral illness with steroids will have any deleterious effects remains to be seen. In view of the usual self-limiting course of IM, steroids at present are recommended only for serious complications.

References

1. Hoagland RJ: The transmission of infectious mononucleosis. Am J Med Sci 229:262-272, 1955.
2. Evans AS: Infectious mononucleosis in University of Wisconsin students. Am J Hyg 71:342, 1960.
3. Evans AS: Infectious mononucleosis in the Armed Forces. Milit Med 135:300-304, 1970.
4. Joint investigation by University Health Physicians and PHLS Laboratories: Infectious mononucleosis and its relationship to EB virus antibody. Br Med J 4:647, 1971.
5. Evans AS, Niederman JC, Sawyer RN: Prospective studies at a group of Yale University freshmen. II Occurrence of acute respiratory infections and rubella. J Infect Dis 123:271, 1971.
6. Hallee TJ, Evans AS, Niederman JC, et al: Infectious mononucleosis at the United States Military Academy. A prospective study of a single class over four years. Yale J Biol Med 3:182-195, 1974.
7. Evans AS, Niederman JC, McCollum RW: Seroepidemiologic studies of infectious mononucleosis with EB virus. N Engl J Med 279:1121-1127, 1968.
8. Diehl V, Henle G, Henle W, et al: Demonstration of herpes group virus in cultures of peripheral leukocytes from patients with infectious mononucleosis. J Virol 2:663-669, 1968.
9. Miller G, Niederman JC, Andrews LL: Prolonged oropharyngeal excretion of Epstein-Barr virus after infectious mononucleosis. N Engl J Med 288:229-232, 1973.
10. Gerber P: Association of EB virus infections with the post-perfusion syndrome. Lancet 1:593, 1969.
11. Hoaglund RJ: The incubation period of infectious mononucleosis. Am J Public Health 54:1699, 1964.
12. Rabuzzi DD: Otolaryngologic aspects of infectious mononucleosis. Can J Otolaryngol 2:329-336, 1973.
13. Chervenick PA: Infectious mononucleosis. DM :1-29 1974.
14. Weary PE, Cole JW III, Hickham LH: Eruptions from ampicillin in patients with infectious mononucleosis. Arch Dermatol 101:86, 1970.
15. Pejme J: Infectious mononucleosis. Acta Med Scand (Suppl) 413:1, 1964.

16. Shuster F, Ognibene AJ: Dissociation of serum bilirubin and alkaline phosphatase in infectious mononucleosis. JAMA 209:267, 1969.
17. Paul JR, Bunnell WW: The presence of heterophil antibodies in infectious mononucleosis. Am J Med Sci 183:90, 1932.
18. Lee CL, Zandrew F, Davidson I: Horse agglutinins in infectious mononucleosis. J Clin Pathol 21:631, 1968.
19. Evans AS, Williams WJ, Beutler E, et al (eds): Hematology, New York, McGraw Hill Book Co, 1922 pp 843-853.
20. Britta W: Diagnosis of infectious mononucleosis by the Monospot Test. Am J Clin Pathol 52:303-308, 1969.
21. Bulden KJ: Corticosteroids in the treatment of infectious mononucleosis. J R Coll Gen Pract 22:87-95, 1972.
22. Bender C: The value of corticosteroids in the treatment of infectious mononucleosis. JAMA 199:529-531, 1967.
23. Proot C, Dalrymple W: A double blind study of eighty-two cases of infectious mononucleosis treated with corticosteroids. J Am Coll Health Assoc 15:62-66, 1966.

SHOULDER PAIN

COL John A. Feagin, Jr., MC

"Shoulder pain" is a complaint frequently heard in the outpatient clinic. Fortunately, it is usually amenable to diagnosis and treatment by the generalist. This is primarily because the shoulder can be readily examined. A nonweight bearing, not tightly constrained joint, it is more "forgiving" than other joints such as the knee.

The objective of this paper is to outline for the non-specialist treating shoulder pain an approach which is safe, provides a high percentage of patient satisfaction, and leaves little margin for error.

The physical examination is more likely to be productive if a differential diagnosis is reached during the history taking. I shall therefore begin with the ingredients of a simple history as a prelude to the differential diagnosis and physical examination.

HISTORY

The history primarily is a careful description of the pain and loss of motion and their effects on the patient's lifestyle. The patient should be asked (1) to describe the pain; (2) whether or not the pain keeps him awake at night; (3) what activities he has had to curtail or give up because of the symptoms; and (4) what medications he is taking to alleviate the pain.

Although the answers to these four questions form the basis for a rational differential diagnosis, the most important by far is that of the character of the pain. Several times during the physical examination the patient should be

queried to ensure that both he and the examiner fully understand the nature of the pain, i.e., continuous, episodic, burning, throbbing; whether or not the pain is activity related; its temporal relationship; its severity and duration; and the effects of position and rest.

DIFFERENTIAL DIAGNOSIS

Although the list of differential diagnoses is extensive, the following three categories should be considered ahead of the others: (1) Tumor; (2) Degenerative disease; and (3) Trauma. A fourth category might be labeled "Miscellaneous". Each of these categories, in turn, has numerous subheadings; however, assigning the patient to one of the first three will inevitably lead to correct diagnosis, treatment, and appropriate referral where necessary.

Tumor

Tumor is a frequent cause of shoulder symptoms and is characterized by unrelenting chronic pain. It awakens the patient at night, is present in the morning, and is unaffected by position or activity. A host of tumors may cause shoulder pain; the one occurring most frequently is Pancoast's tumor of the lung which has a particularly high incidence in smokers.

Degenerative Disease

By age 60, most patients have at least an incomplete degenerative tear of the rotator cuff with associated bursitis. This structure is particularly vulnerable to degenerative lesions because it is subject to impingement beneath the acromial arch and the coracoacromial ligament. Another impingement lesion, bicipital tendonitis, is often difficult to distinguish from subacromial bursitis or an incomplete rotator cuff tear because they have a common nerve supply.

Trauma

The shoulder girdle is mobile and exposed, making it highly vulnerable to injury. Acromioclavicular separation is the most common injury in the young athlete. Instability of the glenohumeral joint is also frequent. Any one of the shoulder girdle joints may be subject to sprain, subluxation, or complete dislocation. These injuries, as well as fractures about the shoulder, rightfully belong in the care of a specialist.

Miscellaneous

Neurovascular entrapment syndrome, referred cervical disc pain, and arterial insufficiency are mentioned for completeness' sake, but they require tests which should be performed by a specialist.

PHYSICAL EXAMINATION

After a careful history eliciting the nature of the pain, the examiner should characterize the order of his differential diagnoses as tumor, degenerative arthropathy, or traumatic instability. Physical examination should then be directed toward the diagnosis which is most suspect. This is best carried out with the patient comfortably seated, disrobed to the waist, with the examiner standing behind the patient where motion can best be evaluated and muscle action observed. The normal shoulder must first be examined as a standard of comparison.

I prefer to examine the shoulder joint in the order shown:

Inspection

Inspection is ideally accomplished with the patient seated comfortably on a rotating stool. A swivel chair may be substituted if the patient sits forward on the chair; this permits turning the patient to examine the right and left sides, front and back. Inspection of the normal and symptomatic sides is quickly accomplished. Careful inspection will demonstrate subtle asymmetry.

Palpation to Elicit Tenderness or Swelling

The supraclavicular fossa is most important to help rule out a Pancoast's tumor. The rotator cuff may be palpated beneath the acromial arch and will be tender if bursitis or significant cuff tear exists. The coracoid, acromioclavicular, scapuloclavicular, axilla and scapulothoracic joints, as well as the cervical and thoracic spines, should all be palpated. The carotid and radial pulses should also be palpated.

Excursion

Range of motion is best tested by asking the patient to do practical things such as scratching behind his head, reaching behind his back, and raising his arm as high as possible as when changing a light bulb. These motions effectively screen for limitations of abduction, internal rotation, and forward elevation.

Two specific tests are the apprehension test and the drop arm test. In performing the apprehension test, an attempt is made to lever the humeral head out of the glenoid; discomfort is usually indicative of an unstable glenohumeral joint (Fig 1). In the drop arm test (Fig 2), the arm is gently placed at 90 degrees of abduction by the examiner. The patient is asked to hold the arm in this position; if he cannot do so, a significant tear of the rotator cuff may be suspected.

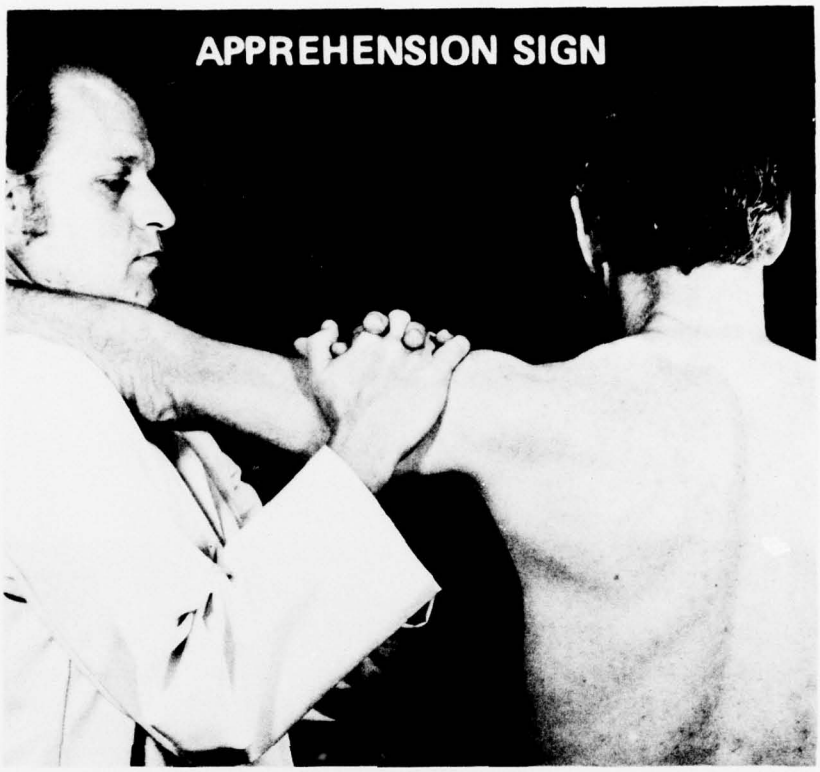


Fig 1. The apprehension sign when positive is indicative of instability of the shoulder.

Neurologic Status

Instruct the patient to perform three simple muscle screening tests in an effort to determine the neuromuscular status:

- Clasp the hands and push them together in front of the body. This tests the anterior deltoid and all the forward flexors of the shoulder girdle including the biceps, pectoralis major, and subscapularis.

- Extend both hands in front of the body and push against a desk or other fixed object. When viewed from the rear, this allows check-

checking of the scapular stabilizers: the trapezius, the rhomboids, the serratus anterior, and the teres major,

- Make a perfect "O" with the thumb and the little finger, thereby tensing the thenar and hypothenar muscles representing C6, C7, and C8 nerve roots.

The above three muscle tests comprehensively check the shoulder girdle and brachial plexus.

Fig 2. The "drop-arm" sign when positive is characteristic of a rotator cuff tear.

The carotid and both radial pulses may be checked while the patient carries the cervical spine through a full range of motion. The radial pulses may also be checked with the arms overhead. There should be little change in the quality of these pulses regardless of position of the neck or arm.

The foregoing physical examination, if well organized, takes only four to five minutes and is quite comprehensive if the examiner is observant and makes full use of his tactile senses.

DIAGNOSTIC TESTS

X-rays are important because tumors are a frequent cause of shoulder pain. The apical lordotic x-ray of the chest is the single most important screening test. This view includes both shoulders, the supraclavicular fossa, the acromioclavicular and scapuloclavicular joints, the cervical spine, the clavicles, scapula, the humeral heads, and ribs. Further x-rays which may be helpful are (1) the humeral head in external and internal rotation for calcific bursitis; (2) the axillary or West Point views for erosion of the glenoid in cases of instability; and (3) a bicipital view for calcification in the biceps tendon.

Arthrography of the shoulder may be indicated if the drop arm test is positive and a significant tear of the rotator cuff is strongly suspected. Surgery must be entertained when the tear is complete. Electromyography is sometimes necessary, particularly when atrophy or weakness is present. Vascular studies may also be indicated but these are best performed by specialists.

TREATMENT

The shoulder can be treated symptomatically, providing the history and physical examination lead to a diagnosis which falls in the area of the generalists, i.e., a degenerative lesion of the shoulder such as bursitis or arthritis. Aspirin is always a good start: 0.60 mg every six hours is an effective analgesic as well as an anti-inflammatory agent. If aspirin does not relieve the pain in 72 hours, injection of the rotator cuff with a long acting steroid is warranted

and simple to administer. This is best done from the posterior using a 1½ inch 21-gauge needle to inject 5 ml of 1 percent xylocaine and 1 ml of the steroid (Fig 3). In subdeltoid bursitis, relief is attained in over 75 percent of cases and the patient is gratified.

Ultrasound about the shoulder, Codman's pendulum exercises, shoulder shrug exercises, and active resistive range of motion exercises are indicated when motion is limited. Frequently a "frozen shoulder" can be brought back to a nearly full range of motion through carefully supervised physical therapy. If the pain is recalcitrant to the simple measures described above, specialty referral is indicated.

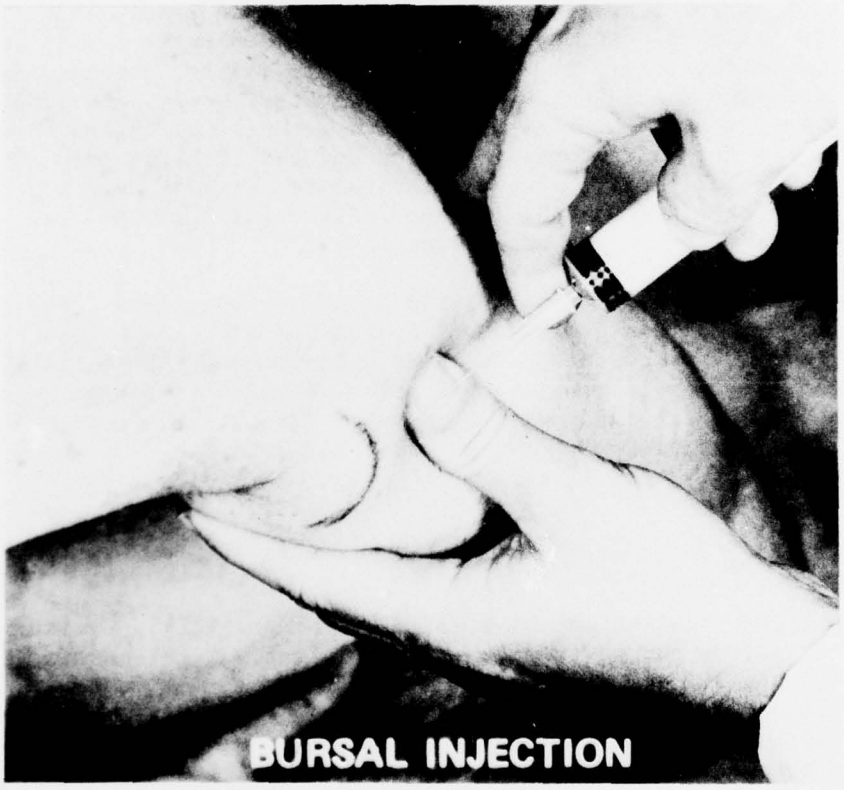


Fig 3. The landmarks for subdeltoid bursal injection are shown from the posterior with the arm in a relaxed position.

FOLLOWUP

Repeat physical examination and reevaluation are indicated when pain persists. Shoulder pain can generally be relieved by injection or analgesics; however, if relief is not prompt, reevaluation is indicated.

CONCLUSION

Management of shoulder pain is a frequent and satisfying challenge to the outpatient physician because (1) physical examination is productive and highly diagnostic; (2) the joint is nonweight bearing and "forgiving"; and (3) simple treatment usually results in relief of symptoms.

References

1. DePalma AF: Surgery of the Shoulder. Philadelphia, Lippincott, 1973, 376 pp.
2. Hoppenfeld S: Physical Examination of the Spine and Extremities. New York, Appleton-Century-Crofts, 1976, 176 pp.
3. Moseley HF: Recurrent Dislocation of the Shoulder. Montreal, McGill University Press, 1961, 126 pp.
4. Moseley HF: The shoulder. Surg Clin North Am 43:1-126, 1963.

ACKNOWLEDGEMENT

Nina Sanders, Technical Publications Editor, Letterman Army Medical Center, and the Assistant Editor, Cathleen Swee, provided the Guest Editor with invaluable help and advice in preparing this symposium. Their expertise, competence, and patience are gratefully acknowledged.

☆U. S. GPO: 1977—792-485